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EPONYMS.

BY SIR D'ARCY POWER, K.B.E., LONDON.

XIX. THOMAS'S HIP SPLINT.

HUGH OWEN THOMAS is a good example of a class of men who are not rare in the profession of medicine. Engaged in a busy general practice, Nature had endowed him with keen observation and with a mechanical genius which he turned to account in the treatment of his patients, untrammelled by any thought of the scientific or pathological principles involved. Nature to him was a personality working always towards a cure, and her efforts were best helped by rest. Thomas devoted all his energies to secure rest. In broken bones, in diseased joints, in obstructed bowels, he immobilized the parts, and by unremitting attention to minute detail he assured himself that the rest was uninterrupted, for he took the absence of pain as his criterion.

He was born at Tyn Llan, Bodedern, in the Isle of Anglesey, on August 3, 1834, whilst his mother was on a visit to her parents, and was the eldest of five boys, all of whom became doctors. His father, Evan Thomas, was a bone-setter practising at 3 Great Crosshill Street, Wallasey, a suburb of Liverpool. He was the seventh in linear descent of a family believed by the neighbours to be endowed with a special gift of healing. Some of the members were well-to-do farmers who bestowed their gift without fee or reward like Cosmas and Damian our patron saints; the poorer members, like Evan Thomas, took money, but as a business it was not lucrative.

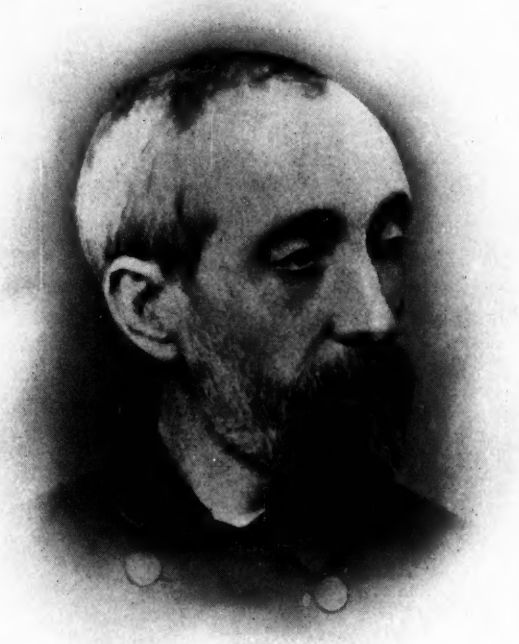
Hugh Owen Thomas, the son, was apprenticed to his maternal uncle, Dr. Owen Roberts, of St. Asaph's, at the age of 17, and as Dr. Roberts was surgeon to the workhouse and infirmary he was fortunately situated. He entered the University of Edinburgh at the age of 22, and had to maintain himself on the allowance of ten shillings a week, which was all that his father gave him. Here he came under the influence of Hughes Bennett, Professor of the Institutes of Medicine, who taught his students to observe precisely and methodically for themselves and to employ all modern methods of precision. Probably at the suggestion of the Rev. Thomas Guthrie, D.D., the great Edinburgh preacher of the day, he became secretary of a temperance

society and maintained its principles during the rest of his life. He left Edinburgh in 1856 without taking a degree, and entered as a medical student at University College in London. He obtained the diploma of M.R.C.S. Eng. in 1857, and immediately went to Paris, where he spent a few weeks and especially admired the skilful work of the French makers of surgical instruments. He then returned to Liverpool to help his father, but the rule-of-thumb methods adopted were uncongenial to his better educated mind, and in 1859 he moved to a small house in Hardy Street which he occupied until 1870, when he moved to 11, Nelson Street. He soon gained a great reputation, first amongst the clubs and industrial societies to which he became attached as medical officer, and afterwards amongst the better classes in the city and its immediate neighbourhood. Money, though useful and necessary, was of secondary importance when weighed against the care of his patients, and he was idolized by the poor of Liverpool, whom he used to treat gratuitously every Sunday morning, although during the remainder of the week some fee was expected and taken. These poor attended his funeral literally in thousands, and showed genuine grief at the loss they had sustained.

A pale, thin man, 5 ft. 4 in. in height, always delicate, he was remarkable for the intelligence shown in his eyes, though the expression of his face was somewhat spoilt by an ectropion resulting from an accidental injury whilst he was a student at Edinburgh. He was an exceptionally keen observer, with a marked appreciation of symmetry or its absence, a trait inherited perhaps from his bone-setting ancestors. A man of the very simplest habits, he never took a holiday, and worked from six in the morning until midnight. His slight figure, closely buttoned in a blue coat, and wearing a naval cap because it had a peak which shaded an eye weakened by an ectropion, was familiar in all parts of Liverpool as he was driven about by a pair of black horses in his home-made gig. He smoked cigarettes almost continuously, and, being of an even temper, he was easy to live with. He was wholly without guile, and was ready to impart his knowledge and show his methods to everyone who sought him. Music was his chief hobby outside the workshop, and his delight was to play the flute whilst his wife accompanied him on the piano. He knew Lucretius almost by heart, and in later life he interested himself in archæology, chiefly Egyptian. He was happily married, though childless. Of outside honours he received but one, the M.D. of the University of St. Louis, which was conferred upon him two years before his death.

Although Thomas is most generally known by the ingenious splints he invented, he was the author of several works which considerably influenced surgical opinions and methods. Amongst these were *Chronic Joint Disease; Compound Fracture of the Lower Jaw; Dislocations; Diseases of Joints; Fractures of the Upper and Lower Extremities; On Hip, Knee, and Ankle; Intestinal Obstruction; New Lithotomy Operation; Nerve Inhibition; Surgical Essays;* and many articles of a polemical character. His special surgical activities were directed to tuberculous arthritis. He preached the doctrine of prolonged and uninterrupted rest, and invented his splints for recumbency and also for early ambulatory exercise. Excision and amputation were almost the routine methods adopted by his contemporaries in the treatment of chronic disease of

the joints. His teaching did much to originate a more conservative treatment, and the methods he practised fifty years ago are those in common use to-day. He introduced many ingenious devices for the cure of fractures, and his patients rarely had more than half an inch of shortening after fracture of the femur. He was amongst the first to use percussion for ununited fractures, and he employed venous congestion long before it was used by Bier. He was, too, the first to insist upon the need for keeping paralysed muscles in a state of relaxation in cases of poliomyelitis. His versatility was shown in his treatment of intestinal obstruction at a time when no operation was



HUGH OWEN THOMAS

performed for its relief. He often kept such patients starved for thirty or forty days, only allowing them a little arrowroot and water, giving hypodermic injections of morphia to check peristalsis and copious draughts of water to percolate through the inflamed intestinal area. He set his face rigidly against enemata. By these means he saved many lives at a time when it was usual to give castor oil and administer turpentine enemata to relieve the obstruction. The Museum of the Royal College of Surgeons of England contains a most interesting case of instruments which he invented for the operation of lithotomy, an operation in the performance of which he was very expert.

Although rough in manner, he gained his results, as well as the confidence of the patients and their friends, by his constant and personal attention to details and by the extreme gentleness of his manipulation. There could, indeed, be no greater proof of the confidence he inspired than his ability to keep a north countryman on a starvation diet for a month. The apparatus he employed was made at home and under his own eyes, and to this end he kept a saddler and a blacksmith in constant employment, whilst his workshop was fitted with the best lathes that money could buy. He died on January 6, 1891, and his practice was carried on by his nephew on the maternal side, my friend Sir Robert Jones, K.B.E., to whom I am indebted for many of the personal facts in this short notice.

The photograph of Mr. Thomas is from one kindly supplied by Sir Robert Jones.

(To be continued.)

THE PATHOGENESIS AND TREATMENT OF TRAUMATIC NEURITIS OF THE ULNAR NERVE IN THE POST-CONDYLAR GROOVE.*

By HARRY PLATT, Manchester.

INTRODUCTION.

When the ulnar nerve reaches the post-condylar groove at the elbow it comes to occupy a position of extreme vulnerability; but it is not the exposed situation of the nerve-trunk which alone determines the incidence of traumatic lesions at this level. Under normal conditions the nerve, though securely anchored in the groove, is also mobile enough to accommodate itself to the repeated changes in tension which occur during the movements of flexion and extension of the elbow-joint. This mechanism is readily disturbed when, as a result of injury, the nerve acquires a limited range of excursion or no longer conforms to the shape or orientation of its bed. In a certain small proportion of individuals the nerve is hypermobile, and, in the position of full flexion of the elbow, slips forward on to the epicondyle. This anatomical peculiarity may pass unnoticed for many years until a complete dislocation occurs, and the frequent repetition of the displacement gives rise to the symptoms of traumatic neuritis.

In the pathogenesis of certain types of ulnar-nerve injury an alteration in the normal relation between the nerve and its bed plays an all-important rôle. Such lesions do not ordinarily embrace the highly destructive primary injuries of this nerve which result from gunshot wounds or other traumata equally grave, although this factor may operate in some measure even in these conditions.

The special injuries which alone form the subject of this paper may be divided somewhat arbitrarily into three clinical groups: (1) *Ulnar-nerve lesions associated with recent fractures of the lower end of the humerus*; (2) *Late ulnar-nerve involvement after fractures*; and (3) *Recurring dislocation of the ulnar nerve.* In all three groups the actual nerve injuries belong for the most part to the class of incomplete lesions, and may thus be included under the broad title of traumatic neuritis.

1. ULNAR-NERVE LESIONS ASSOCIATED WITH RECENT FRACTURES OF THE LOWER END OF THE HUMERUS.

THE INCIDENCE OF THE NERVE LESIONS.

Considered as a whole, the ordinary fractures of civil life are but rarely complicated by the presence of injuries of peripheral nerves. In certain

* From the Orthopædic Service, Ancoats Hospital, Manchester.

regions, however, a combination of the two lesions provides a clinical picture which is by no means unfamiliar. The anatomical distribution of these dual injuries is shown in a recent paper by Dean Lewis and Miller,[1] of Chicago, who have collected 239 examples from surgical literature and from their own hospital records. The figures of these writers indicate that 80 per cent of the cases are fractures of the *humerus*, the common site of the fracture being in the lower third of this bone (60 per cent). Of the peripheral nerves implicated, the musculospiral accounts for 60 per cent, and the *ulnar nerve* for 25 per cent of the cases.

One of the earliest and most important investigations on this subject appeared in 1899 in the form of an admirable study by Broca and Mouchet,[2] of Paris, of the nerve lesions complicating certain fractures of the lower end of the humerus: 78 consecutive fractures of this type were observed during a period of two years, and in 9 of them a nerve injury was recorded. On close analysis these fractures were divided into two groups: (*a*) Fractures of the *external condyle*, of which there were 40 examples; and (*b*) *Supracondylar* fractures, in the remaining 38. In the former group there were 3 nerve injuries, and in the latter 6. In several cases the nerve lesions were multiple. The nerve-trunks involved were the ulnar (5 cases), median (5 cases), and the musculospiral (3 cases). It is interesting to note that during the same period in the practice of the authors there were no nerve injuries accompanying dislocations of the elbow-joint, other types of fracture of the lower end of the humerus, fractures of the olecranon, or fractures of the head of the radius.

In this paper of Broca and Mouchet the pathogenesis of the nerve injuries was considered in some detail. Special emphasis was laid on the chronology of the lesions, and a distinction made between *primary*, *secondary*, and *tardy* injuries, the latter term being applied to what had long been recognized as a clinical entity, viz., the late involvement of the ulnar nerve after fractures sustained in early childhood. Mouchet himself had already drawn attention to this delayed type of ulnar-nerve lesion in his Paris thesis submitted in 1898, and had realized that the injury which preceded it was almost invariably a fracture of the *external condyle*. Since that date, Mouchet has closely identified himself with this subject in a number of valuable contributions, and most notably in a comprehensive paper which appeared in the *Journal de Chirurgie* in 1914.[3]

The attention which has been focused in recent years on this interesting but relatively uncommon form of late ulnar-nerve lesion has tended perhaps to obscure the significance of the occasional involvement of this nerve in recent fractures of the elbow region. My own interest in this subject was first aroused a few years ago by the discovery, from time to time, of ulnar-nerve lesions amongst the recent elbow fractures treated in the fracture clinic of my surgical unit. Whilst it was obvious at once that the nerve injury was usually a transitory clinical phenomenon, in a few cases the interference with the conduction in the nerve was sufficiently grave to merit serious consideration. With a view to the acquisition of knowledge regarding the incidence and prognosis of these dual lesions in ordinary routine fracture treatment, a survey was made of the elbow fractures dealt with in this clinic over a four-year

period, viz., 1921 to the end of 1924. The number of such fractures under treatment was 329, and they were classified as follows :—*

Humerus.—Supracondylar fractures	..	95
Fractures of the internal condyle	..	74
Fractures of the external condyle	..	83
Olecranon	..	38
Radius.—Head and neck	..	39
Total	..	329

In this series, 9 fractures (*Series I*)—all involving the *humerus*—were accompanied by a nerve injury, the nerve affected in each case being the *ulnar*. The absence of nerve lesions in the olecranon and radius fractures, and the immunity of the median and musculospiral nerves in this series, are facts of interest, but they do not demand special comment here.

Eight of the nerve lesions accompanied fractures of the *internal condyle*, and *one* a supracondylar fracture. The fractures were all of the 'closed' (simple) type. In *seven* cases spontaneous recovery was seen, full conductivity being restored in periods varying from three to eight months. In *one* case, however, at the end of nine months the clinical signs of a severe neuritis still persisted, and for this reason exploration of the nerve was undertaken. Following the operation of displacement of the nerve in front of the internal condyle rapid recovery ensued. The remaining patient, who presented the clinical signs of a very mild nerve lesion, ceased to attend the clinic after the first few weeks, but it is not unreasonable to assume that full recovery occurred.

If the group of fractures quoted here may be regarded as a typical consecutive series, it is evident that involvement of the ulnar nerve is an infrequent complication of fractures of the lower end of the humerus (9 in 252 fractures—roughly 3·7 per cent). A brief résumé of the clinical histories of these complicated injuries will now be presented before we embark on a more extended inquiry into the various factors which may be concerned in the pathogenesis of such nerve lesions.

RECORDS OF CASES.

Series I. Cases 1–9.

Case 1.—**H. B., age 13 (1921). Fracture of internal condyle (fracture involving the joint surface).**

Ulnar-nerve Involvement.—Signs of complete nerve-block noticed at the end of four weeks.

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* The classification adopted here for fractures of the lower end of the humerus is a simple one, which has proved convenient in the routine work of a fracture clinic. Each variety includes a number of subdivisions. Thus in the *supracondylar* group there will be a few uncommon or rare types, such as the T-shaped fracture and the diacondylar fracture. The *internal condyle fractures* include both fractures of the whole condyle and fractures limited to the epicondyle. Similarly, under the heading of *external condyle fractures* are included such rare injuries as isolated fracture of the capitellum.

It must also be explained that the fractures in the above series were received in the clinic direct from the casualty department of the hospital. The figures do not include complicated fractures treated elsewhere in the initial stage and referred for a consultation at a later date.

Neurological Syndrome.—Wasting with paralysis of the interossei, adductor pollicis, and hypothenar muscles; complete sensory loss. Traumatic ulceration on the dorsum of the little finger.

SUBSEQUENT COURSE.—

Result of the Nerve Injury.—Rapid spontaneous recovery. No sign of involvement three years later.

Result of the Fracture.—Full mobility of the elbow; no increase in the carrying angle. The internal condyle is thickened and the nerve is placed more superficially.

Case 2.—**V. C., age 12 (1922). Fracture of internal condyle (fracture involving the joint surface).**

ULNAR-NERVE INVOLVEMENT.—Signs of an incomplete nerve-block were noted at the end of three weeks.

Neurological Syndrome.—Flattening of the hypothenar eminence and interosseous spaces, with paresis in these muscles; analgesia in the ulnar area of the hand.

SUBSEQUENT COURSE.—

Result of the Nerve Injury.—Spontaneous recovery with almost complete restoration of function at the end of eight months. At the end of sixteen months no trace of any interference in the conductivity of this nerve.

Result of the Fracture.—Full range of flexion, but extension falls short of the full range by 10°. Moderate degree of cubitus valgus deformity.

Case 3.—**A. B., age 12 (1922). Fracture of internal condyle (epicondyle only).**

ULNAR-NERVE INVOLVEMENT.—Incomplete loss of conduction noted at the end of three weeks.

Neurological Syndrome.—Analgesia of the little finger, with paresis and flattening of the hypothenar muscles.

SUBSEQUENT COURSE.—

Result of the Nerve Injury.—Rapid spontaneous recovery, complete at the end of four months.

Result of the Fracture.—Full restoration of mobility of the elbow, with no increase in the carrying angle.

Case 4.—**E. G., age 14 (1922). Fracture of internal condyle (fracture involving the joint surface).**

ULNAR-NERVE INVOLVEMENT.—Signs of an incomplete block noted at the end of four weeks.

Neurological Syndrome.—Flattening and paresis of the hypothenar muscles, with full ulnar sensory loss.

SUBSEQUENT COURSE.—

Result of the Nerve Injury.—Spontaneous recovery with complete restoration of conduction at the end of eight months.

Result of the Fracture.—Full range of mobility of the elbow-joint with a normal carrying angle. The internal condyle is thickened and the ulnar nerve appears to be more superficial than on the opposite side, but is not unduly tender.

Case 5.—**A. H., age 16 (1922). Fracture of the internal condyle (fracture involving the joint surface).**

ULNAR-NERVE INVOLVEMENT.—Signs of an incomplete nerve-block noted at the end of two weeks.

Neurological Syndrome.—Analgesia in the ulnar area, but no appreciable interference with the motor power in the ulnar intrinsic muscles.

SUBSEQUENT COURSE.—The patient ceased to attend the clinic at the end of six weeks; at that time the elbow-joint had regained full mobility. It is presumed that the nerve lesion recovered completely. Repeated efforts to trace the patient have been unsuccessful.

Case 6.—**F. B., age 12 (1922). Fracture of internal condyle (epicondyle only).**

ULNAR-NERVE INVOLVEMENT.—Signs of an incomplete nerve-block noted at the end of three weeks.

Neurological Syndrome.—Analgesia in the ulnar area, with paresis and wasting of the hypothenar and interossei muscles.

SUBSEQUENT COURSE.—

Result of the Nerve Injury.—Spontaneous recovery with complete restoration of conduction at the end of six months.

Result of the Fracture.—Full range of mobility at the elbow with no alteration in the carrying angle.

Case 7.—**W. H., age 12 (1922). Fracture of internal condyle (fracture involving the joint surface).**

ULNAR-NERVE INVOLVEMENT.—Signs of an incomplete nerve-block noted at the end of three weeks.

Neurological Syndrome.—Marked paræsthesia, with an incomplete sensory loss; wasting and paresis of the interossei and hypothenar muscles.

SUBSEQUENT COURSE.—

Result of the Nerve Injury.—Condition nine months later: All the signs of a severe ulnar neuritis. The nerve-trunk is exquisitely tender on palpation, and in full flexion of the elbow is abnormally superficial. There is complete paralysis of all the ulnar intrinsic muscles, with marked tenderness of the hypothenar eminence. Moderate clawing of the little and ring fingers is present, and there is an incomplete sensory loss. Operative exploration of the nerve considered advisable.

Result of the Fracture.—The elbow at this date allows the full range of flexion, and practically a complete range of extension. There is a definite increase in the carrying angle.

OPERATION.—Neurolysis with anterior transposition. The nerve in the region of the groove showed a restricted mobility, and was slightly thickened, the area of the lesion being represented by an early fusiform neuroma. The nerve was freed, displaced in front of the internal condyle, and buried in an intramuscular bed.

Rapid subsidence of the signs of irritation, with steady return of conduction.

Condition eighteen months after the operation: Complete restoration of conduction in the ulnar nerve, with no appreciable impairment of function.

Case 8.—**C. S., age 8 (1924). Fracture of internal condyle, complicating a posterior displacement of the elbow-joint.**

After reduction of the dislocation, the elbow was rested in the position of full flexion for two weeks. Active movements and massage followed.

ULNAR-NERVE INVOLVEMENT.—Signs of incomplete loss of conduction noted at the end of six weeks.

Neurological Syndrome.—Very slight flattening of the muscles bellies of the flexor carpi ulnaris, hypothenar eminence, and interossei. No appreciable paresis; no trace of sensory loss. The ulnar nerve at the elbow is slightly more superficial than on the opposite side.

SUBSEQUENT COURSE.—

Result of the Nerve Injury.—Complete recovery of condition and function eight months later.

Result of the Fracture-dislocation.—Full range of mobility in the elbow-joint was restored. The internal condyle remains thickened and irregular.

Case 9.—**J. P., age 5 (1923). Supracondylar fracture.**

Considerable swelling in the early stages; backward displacement of the lower end of the diaphysis. Further readjustment of the fracture was necessary two weeks after the injury, and this was carried out under an anæsthetic.

ULNAR-NERVE INVOLVEMENT.—Signs of an incomplete interruption of conduction in the ulnar nerve were noted at the end of six weeks.

Neurological Syndrome.—Wasting and paralysis of the hypothenar muscles and hypo-æsthesia in the ulnar skin area; slight tendency to clawing of the little finger;

marked tenderness of the hypothenar muscles. Nerve-trunk at elbow definitely tender, but not palpably thickened.

SUBSEQUENT COURSE.—

Result of the Nerve Injury.—Spontaneous recovery with practically complete restoration of function at the end of eight months.

Result of the Fracture.—Full mobility of the elbow-joint, with a barely appreciable increase in the carrying angle.

To the above may now be added, for comparative purposes, three further examples (*Series II*) of severe ulnar-nerve lesions complicating (*a*) an internal condyle fracture, and (*b*) two cases of posterior dislocation of the elbow-joint. These patients had received treatment in the early stages elsewhere, and came under observation after several weeks had elapsed from the date of the injury. In two cases it was considered advisable to explore the injured nerve.

SERIES II. CASES 10-12.

Case 10.—**T. C., age 20 (1924). Fracture of internal condyle, complicated by a small chip fracture of the coronoid process of the ulna.**

Patient was seen after eight weeks' treatment elsewhere. The elbow had been fixed in full flexion for ten days, and at the end of that period *energetic passive* movements were begun. *Involvement of the ulnar nerve* was recognized about the end of the fourth week.

CONDITION WHEN FIRST SEEN.—The elbow showed considerable distortion in the region of the internal condyle, and there was marked limitation of the range of mobility of the joint. There was an obvious ulnar-nerve lesion, with all the signs of a complete block. In view of the syndrome, and the experience of previous cases, exploration of the nerve-trunk was considered advisable.

OPERATION.—The ulnar nerve was exposed from the lower third of the upper arm to the junction of the upper and middle thirds of the forearm. There was considerable scarring of the aponeurosis and muscle-fibres of the common flexor origin from the internal condyle. The nerve-trunk was intact, but in the region of the ulnar groove was extremely attenuated and reduced to a narrow flat ribbon over a length of one inch. The area of the lesion was demarcated above and below by a small neuroma involving the whole thickness of the nerve-trunk. Complete resection of the area of the lesion was carried out, and *end-to-end suture* performed, the sutured nerve being *displaced* in front of the internal condyle and embedded in the usual way.

Case 11.—**C. M., age 15 (1924). Posterior dislocation of elbow-joint.**

This patient also was treated elsewhere in the early stages. After reduction of the dislocation, the elbow had been rested for ten days and *forcible passive movements* begun at the end of that time. The ulnar-nerve lesion was recognized about the end of the fifth week.

CONDITION WHEN FIRST SEEN (thirteen weeks subsequent to the injury).—The elbow-joint showed practically an undiminished range. There was an ulnar-nerve involvement, with all the signs of a severe but incomplete block. Exploration of the nerve-trunk advocated.

OPERATION.—The ulnar nerve was exposed in the lower third of the upper arm as far as the junction of the upper and middle thirds of the forearm. In the whole area exposed the nerve-trunk was found to be anchored to its bed by thin filmy strands, so that its mobility was very considerably restricted. This abnormal anchorage was best marked in the groove. The sheath of the nerve showed many points of vascular injection, and in the region of the groove the trunk was reduced to a calibre representing about one-half of the normal cross-section. There was no definite bulb, but there was an appreciable amount of induration at the upper limit of the attenuated segment. It was noted also that the fibres of origin of the superficial flexor muscles from the internal condyle contained a good deal of tough scar

tissue. The nerve-trunk was *displaced* in front of the internal condyle and buried in the usual fashion.

Case 12.—J. D., age 16 (1920). **Posterior dislocation of elbow-joint.**

The patient was seen three weeks after the injury. Two unsuccessful efforts had already been made to reduce the dislocation, and *repeated forced movements* had been utilized in an attempt to reproduce mobility.

On Examination.—The left elbow showed an extensive swelling and was completely fixed in the position of right-angle flexion. Palpation of the bony points suggested that the dislocation still existed. This view was confirmed by radiograms. All the signs of a *complete ulnar-nerve block* were present.

A further attempt was made to reduce the displacement by manipulation alone, but this failed, and accordingly an open operation was performed. It was found necessary to excise the lower end of the humerus. The ulnar nerve was not exposed at the time.

Subsequent Course.—

Result of the Nerve Injury.—Steady spontaneous recovery was seen, and for this reason the nerve-trunk was not explored. Four years from the time of the injury no clinical signs of ulnar-nerve involvement could be demonstrated.

Result of the Dislocation (excision of the elbow).—A distorted elbow, allowing full flexion and almost complete extension, with adequate stability.

PATHOGENESIS.

It is desirable first of all to define the chronological relation between the fracture and the nerve lesion, for if the nerve injury is produced at the same moment as the fracture—a primary lesion—the incidence of this complication is beyond the control of the surgeon. But if it is clearly proved that the clinical signs of involvement of the nerve-trunk have arisen *de novo* in the first few weeks after the injury—a *secondary* lesion—the methods employed in the early treatment of the fracture must be subjected to critical scrutiny.

Secondary nerve lesions are usually regarded as due to the compression effect of callus or to the continued pressure of an unreduced bony fragment. If by callus-compression is meant the actual incorporation of a nerve-trunk in newly formed fibro-osseous tissue, then in my opinion this is an extremely rare cause of nerve injury associated with fractures. In more than 600 personal operations on injured nerves, complete ensheathment of a nerve in callus has been seen twice only. But exuberant callus formation may easily produce a distortion of the nerve-bed, and so place the nerve under conditions of abnormal tension or friction. Under such circumstances a traumatic neuritis may be excited by the ordinary movements of the neighbouring joint. The kinking, or the gradual laceration of a nerve-trunk over a bony projection, is a well-recognized cause of nerve injury, and one which has been demonstrated on many occasions during exploratory operations.

In the treatment of fractures of the elbow region both these potential etiological factors are almost entirely under the control of the surgeon. Thus it is common knowledge that excessive callus formation is almost invariably the result of the injudicious use of forced movements of the elbow-joint in the early stages. It is also obvious that, with an early and efficient reduction of any bony displacement, the risk of a secondary nerve injury due to this cause is negligible. But even when such definite gross therapeutic errors are

excluded, it is clear that there are occasional fractures in which a secondary nerve lesion must be accepted as an inevitable complication.

The chronological definition of any given nerve injury must often be based on careful clinical observation. It is generally taught that the syndromes of a primary and a secondary nerve lesion present certain differences. In the former the onset of the signs of nerve-block is abrupt, and the loss of conduction rapidly becomes complete. In the latter the onset is more gradual, and the signs of block deepen slowly and may remain incomplete for an indefinite period. But in actual practice these distinctions are not always clear-cut, and from clinical records alone it is by no means easy to assign a nerve injury to its appropriate chronological group. This difficulty is illustrated when we attempt to classify the individual cases in the first series quoted above (*Cases* 1–9). It is seen here that in no case was the syndrome of a nerve lesion identified before the third or fourth week; and further, in most of the patients the signs of an incomplete nerve-block only were present. At first sight these facts would appear to support the view that the lesions were all of the secondary type. In this assumption, however, there lies a possible fallacy, for during the first two or three weeks the local signs of bony trauma dominate the clinical picture, and a peripheral nerve lesion may easily escape notice, even though that lesion dates from the time of the injury. This applies exclusively to incomplete lesions of the *ulnar* and *median* nerves; the wrist-drop of the *musculospiral* palsy, on the other hand, is rarely missed at the onset.

The nerve injuries in the first series of cases, in my judgement, belong to the *primary* class. In the *internal condyle* fractures this view seems inconestable when the mechanism of the production of the fracture is reviewed, for it is evident that the nerve sustains a direct contusion at the moment the condyle is chipped off. Moreover, as the separated condyle is usually drawn forwards away from the nerve by the pull of the flexor muscles, the local conditions do not ordinarily favour the production of a secondary lesion if the early treatment is on correct lines. *Case* 9, a *supracondylar* fracture, is perhaps more difficult to place, but may not unreasonably be classified as a primary lesion.

In the second series similar difficulties arise, but on the whole it seems fair to allocate these lesions also to the primary class.

This decision, however, does not warrant the assumption that either the severity of the lesion or the fate of the injured nerve is determined alone by the primary trauma. In the type of nerve injury under consideration, the distinction between primary and secondary lesions is really of little moment. For it is quite clear that during the early stages of treatment a mild primary contusion may be converted into a severe progressive neuritis. A primary lesion may then pass into the secondary class. This sequence of events is well illustrated in certain cases in both series recorded above.

In *Case* 7 of the first series—the one example of a severe and persistent neuritis—the early treatment may justifiably be absolved from any direct share in the production of the nerve lesion. The elbow was treated from the beginning in accordance with the routine for all such fractures, viz., rest in full flexion for a period of at least two weeks, followed by very gradual

mobilization of the joint by means of *active* movements only. When the signs of ulnar-nerve irritation became apparent, efforts to reproduce joint mobility were suspended. In spite of this, the intensity of the neuritis, as measured both by subjective and objective signs, deepened steadily. Here was a severe primary contusion of the nerve associated with a slight disturbance of the nerve-bed due to the fracture. In consequence the swollen nerve became more superficially placed, and its range of excursion in the groove was curtailed. In addition, at the end of a few months the elbow showed a mild cubitus valgus deformity and the full range of extension could not be obtained. In short, a combination of factors capable of producing a friction or tension neuritis in the anchored nerve. The secondary neuritis in this case, in my opinion, was an unavoidable complication. The dramatic effect of the operation of anterior transposition of the nerve adds further testimony to this conception of the pathogenesis of the nerve injury.

In the remaining cases, which were all treated in the routine fashion, the factors described above did not intervene, and the nerve lesions recovered speedily.

In the second series we find three primary nerve lesions in which serious additional damage was inflicted in the first few weeks by injudicious treatment, i.e., the use of repeated *forced passive* movements. The clinical picture in these cases again illustrates the evolution of a friction or tension neuritis; but here the gravity of the lesions was determined by therapeutic mistakes.

It is important to recognize that in all the cases recorded above the harmful effect of forced movements was not dependent on excessive callus formation. Indeed, where exuberant callus and a nerve lesion co-exist, both complications may be produced by a common etiological factor.

SUMMARY.

1. Involvement of the ulnar nerve is a rare complication in recent fractures of the lower end of the humerus (9 in 252 consecutive cases in the writer's series). The common type of fracture to be accompanied by a lesion of this nerve is separation of the *internal condyle*.

2. The nerve lesion is usually a primary contusion—a physiological block without loss of continuity. The symptoms are slight, and rapid spontaneous recovery tends to occur.

3. In exceptional cases the *primary* neuritis may increase in severity during the early stages of treatment, and the lesion may pass etiologically into the *secondary* class.

4. A *secondary* ulnar neuritis of this type may be an unavoidable sequela. In such cases a friction or tension neuritis is produced as a result of a disturbance of the normal relation between the nerve and its bed.

5. A *secondary* neuritis may also be induced by injudicious treatment, i.e., the use of forced passive movements of the elbow in the first few weeks after the injury. Under similar circumstances a secondary neuritis may arise *de novo*.

6. In all cases of severe and persistent neuritis the nerve-trunk should be explored and displaced to a new bed in front of the internal condyle.

2. LATE ULNAR-NERVE INVOLVEMENT AFTER FRACTURES.

It is believed that Duchenne was familiar with this condition, but as far as can be ascertained it was first described as a clinical entity by Panas[4] in 1878. Prior to 1900, cases were recorded by Bowlby, Weber, Guillemain and Mailly, and others; and to this period belongs the first of Mouchet's sequence of illuminating papers. Since that time the subject has received considerable attention from both surgeons and neurologists. Prominent in the more recent literature are the contributions of Sherren,[5] Ramsay Hunt,[6] Adson[7] of the Mayo Clinic, Buzzard,[8] Lusena,[9] Dean Lewis and Miller (loc. cit.).*

The broad clinical picture, which is a characteristic one, has long been recognized. In an adult who has sustained a fracture of the lower end of the humerus in early childhood, the subjective and objective signs of neuritis of the ulnar nerve arise insidiously. The neuritis is slowly progressive, and after a time the nerve in the region of the elbow becomes thickened, and may show a distinct fusiform neuroma (nerve spindle). Many of the earlier cases were regarded as examples of compression neuritis due to the involvement of the nerve in old 'callus', and this idea, though early shown by Mouchet to be illogical, dominated the operative treatment of the lesion for many years. Various types of humerus fracture have been described as antedating this late form of nerve lesion. In 43 cases collected by Dean Lewis and Miller, the primary bony injury was as follows: external condyle fractures, 23; internal condyle fractures, 10; supracondylar fractures, 8; 'epiphyseal separation', 2. Late ulnar palsy has also been described in association with an old infective arthritis of the elbow-joint (Weber, Sherren).

It is important to remember, however, that many of the earlier cases were recorded either before the advent of radiography or at a time when radiographic technique was admittedly imperfect. For this reason the existing descriptions of the bony injury are often unreliable; in a number the type of fracture has been rather a matter of clinical conjecture. This, as we shall see, is a consideration of some importance in connection with the pathogenesis of the nerve lesion. Mouchet's original case followed a fracture of the *external condyle*, and by 1914 he had dealt with seven cases, all corresponding to this one clinical type. (Since 1914 he has seen three additional cases.) In his view, the classical late ulnar-nerve lesion is unlikely to be associated with the other varieties of humerus fracture, because the development of the neuritis is determined primarily by the existence of a gross cubitus valgus deformity. Now this deformation—in an extreme degree—is a characteristic sequela of the complete external condylar fracture and no other, although an inconspicuous increase in the carrying angle is not infrequently present after supracondylar or even internal condyle fractures. It may be said at once that a scrutiny of the cases reported in the last decade, accompanied by full radiographic evidence, bears out the accuracy of Mouchet's conception, i.e., that the late ulnar-nerve lesion is ordinarily a sequela of fractures of the external condyle. This does not preclude the possibility of late involvement occurring after other types of humerus fracture, but such cases are quite exceptional.

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* Since the greater part of this paper was written some two years ago, further important contributions have been made both by Mouchet[10] and Miller.[11]

With this brief introduction we may now proceed to consider in greater detail the clinical features, pathogenesis, and surgical treatment of late ulnar palsy. It will be convenient at this stage to interpolate the clinical records of a series of personal cases (*Series III*) which will serve to illustrate and emphasize many points in the description which follows.

RECORDS OF CASES.

Series III. Cases 13–18.

Case 13.—**Miss R., age 59. Old fracture of external condyle of left humerus. Late involvement of ulnar nerve.**

History.—At the age of 5 sustained a fracture of the lower end of the humerus which left a deformity of the elbow and some limitation of mobility. *Fifty-one years after* the injury, began to experience pain and tingling in the little and ring fingers. Two years after the onset of these symptoms the patient sustained a contusion of the elbow, and from this time onwards her condition became steadily worse. The pain was evoked during the movements of flexion of the elbow and supination of the forearm.

Condition on Examination.—The left elbow showed a marked cubitus valgus deformity with irregularity of both condyles. Movements of the joint were restricted, extension falling short by about one-third of the full range, but flexion was almost complete. At the elbow the ulnar nerve was unduly palpable, and there was an appreciable degree of thickening of the trunk, which was also exquisitely tender.

Neurological Syndrome.—

a. Motor function: The signs of an incomplete nerve-block were present. There was paresis and wasting in the belly of the flexor carpi ulnaris and inner half of the flexor profundus digitorum; but the ulnar intrinsic muscles of the hand were all acting, and there was no visible atrophy.

b. Sensory function: Definite hypo-æsthesia in the ulnar area of the hand, but the protopathic response was normal. There were no vaso-motor paralysis or trophic changes.

A radiogram (*Fig.* 270) showed an old fracture of the external condyle of the humerus with non-union.

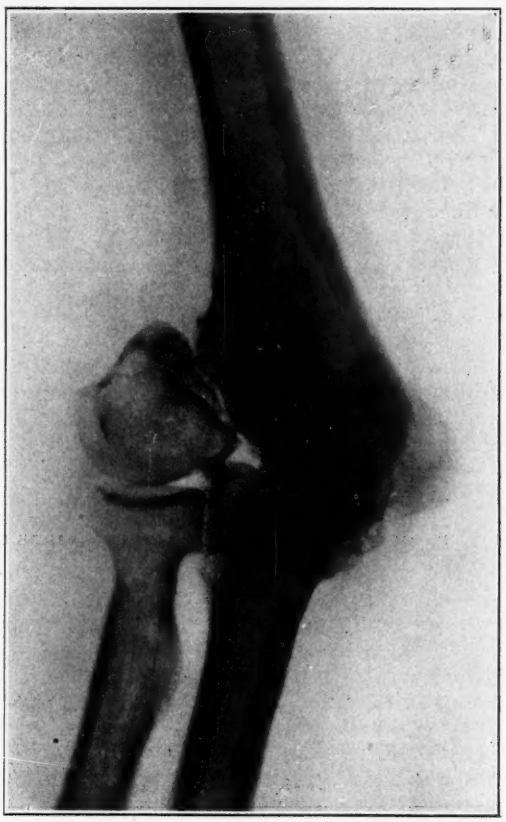

Fig. 270.—*Case* 13. Radiogram of elbow. Late ulnar palsy. Shows: (*a*) The old fracture of the external condyle, with no sign of bony continuity fifty-three years later; (*b*) The apparent overgrowth of the internal condyle; (*c*) The cubitus valgus deformity.

Operation.—Feb. 20, 1921. *Exploration of the ulnar nerve* in the region of the elbow, with *anterior displacement*. In the region of the post-condylar groove the nerve-trunk was found to be slightly swollen, and where lying in contact with the joint capsule a tiny opening was seen which led into the joint cavity. The

cartilaginous covering of the humerus at this spot showed some irregularity, producing a nodular surface. On the deeper aspect of the nerve at this level the sheath was frayed through at one spot; elsewhere the nerve-sheath was thickened, and there appeared to be a slight degree of intraneural induration.

The nerve-trunk was freed in the lower part of the upper arm and upper part of the forearm, and was then displaced in front of the internal condyle, the proximal branches being preserved intact. The nerve was buried in an intramuscular bed.

SUBSEQUENT COURSE.—The operation was followed within a few weeks by complete cessation of the pain experienced during movements of the elbow. Three years later, all the subjective sensory symptoms had completely disappeared, except that the patient was still conscious of a slight numbness in the ulnar distribution of the hand. A full neurological examination at this date was not available.

Case 14.—**Miss N., age 27. Old fracture of external condyle of left humerus. Late involvement of ulnar nerve.**

HISTORY.—At the age of 3 sustained a fracture of the lower end of the humerus. As a result, the elbow-joint had always been deformed and there had never been a complete range of movement. The patient, who was now a professional violinist, during the past few months had become conscious of the development of progressive weakness in the left hand, and had experienced attacks of pain referred along the little finger. She had also noticed a certain amount of wasting of the finer muscles of the hand. In consequence she found increasing difficulty in continuing to play her instrument. (Onset of symptoms twenty-four years after the injury.)

CONDITION ON EXAMINATION.—The left elbow showed a marked deformation with a great increase in the carrying angle, and an undue projection or apparent overgrowth of the internal condyle. There was a full range of flexion, but extension fell short by about 20°. The ulnar nerve was easily palpable in the postcondylar groove, but did not feel enlarged. It was somewhat tender on compression, but this sign was not conspicuous.

Neurological Syndrome.—

a. Motor function: In the hand there was definite atrophy of the interossei, adductores pollicis, and hypothenar eminence. There was marked weakness in the adductor pollicis, interossei, and abductor minimi digiti; the flexor brevis pollicis showed a complete palsy.

b. Sensory function: There was diminished acuity of sensation in the ulnar area, both to light touch and painful stimuli.

A radiogram showed an old fracture of the *external condyle* with fibrous union.

OPERATION.—Nov. 4, 1922. Exploration of ulnar nerve; anterior transposition. In the region of the post-condylar groove the nerve was very slightly swollen and showed an appreciable degree of intraneural induration. The nerve-trunk was freed in the usual way, displaced forwards, and buried in an intramuscular bed.

SUBSEQUENT COURSE.—Within a few weeks after the operation definite and progressive signs of regeneration set in. The patient resumed her violin playing four months later, and eleven months from the time of the operation was doing full duty in an orchestra.

Condition Two Years after the Operation.—There was no trace of wasting of the ulnar intrinsic muscles, and sensory tests showed that there was a mere trace of hypo-æsthesia over the terminal phalanx of the little finger.

Case 15.—**Miss D., age 33. Old fracture of external condyle of humerus. Late involvement of ulnar nerve.**

HISTORY.—At the age of 2 sustained a fracture of the lower end of the left humerus. Perfect movement was restored in the joint, but in later childhood the elbow was noticed to be deformed. Two years ago (thirty-one years after the injury) the patient became conscious of an increasing loss of power in the left hand, particularly marked during piano playing, and also when handling a golf club. Later she began to have attacks of tingling along the inner border of the hand and little finger, and more recently felt that this region was numb. She had found that relief from the subjective symptoms could be obtained by pressing on the ulnar nerve at the elbow.

CONDITION ON FIRST EXAMINATION.—The elbow showed a marked cubitus valgus deformity, the carrying angle being increased by about 30° beyond the normal. The region of the *internal condyle* was unduly prominent, and the whole of the lower end of the humerus appeared to be broadened. The normal relation of the bony points was distorted owing to the obliquity of the lower articular surface of the humerus. The post-condylar groove was shallow, and the ulnar nerve more palpable than usual. The nerve-trunk was obviously thickened, with the formation of a very slight 'spindle'; it was exquisitely tender, and on palpation there was marked tingling referred along the ulnar border of the hand.

Radiograms (*Fig.* 271) showed an old fracture of the external condyle.

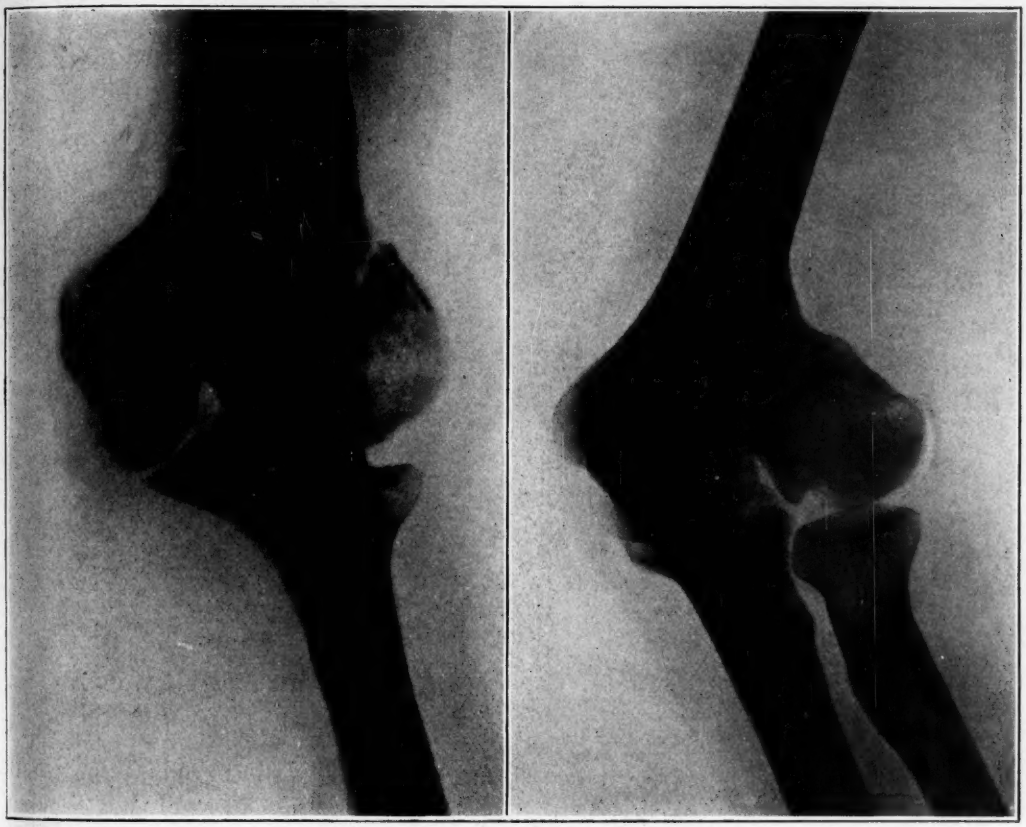

FIG. 271.—*Case* 15. Radiograms of elbow, anteroposterior and semilateral views. Late ulnar palsy. Shows: (*a*) The old fracture of the external condyle; (*b*) The distorted internal condyle; and (*c*) The cubitus valgus deformity.

Neurological Syndrome.—

a. Motor function: Marked atrophy in all the ulnar intrinsic muscles of the hand, with complete loss of voluntary power. The flexor carpi ulnaris and flexor profundus digitorum (inner half) were acting, but were definitely weak.

b. Sensory function: There was definite hypo-æsthesia and incomplete loss of protopathic sensation in the ulnar area of the hand. The deep sensation was normal. There were no conspicuous vasomotor changes, and no trophic signs.

OPERATION.—March 21, 1924. Anterior transposition of the ulnar nerve. Nerve-trunk in the groove was slightly thickened; sheath intact.

SUBSEQUENT COURSE.—Complete disappearance of the paræsthesia signs two months after the operation.

Case 16.—**Mrs. B., age 32. Old fracture of left external condyle. Late involvement of ulnar nerve.**

HISTORY.—At the age of 6 sustained a fracture of the left elbow; a well-marked deformity had been present since the year of the injury, and the elbow-joint had never recovered full mobility. Four years ago (twenty-two years after the accident) the patient began to have attacks of pain radiating from the inner side of the elbow to the little finger. She had worked for many years in a cotton mill, duties which involved constantly repeated flexion and extension of the elbow. Shortly after the onset of pain, wasting of the small muscles of the left hand became apparent. At the time of examination, although still engaged in her occupation, the patient had begun to find it almost impossible to continue at work owing to the severe pain and the progressive weakness in the hand.

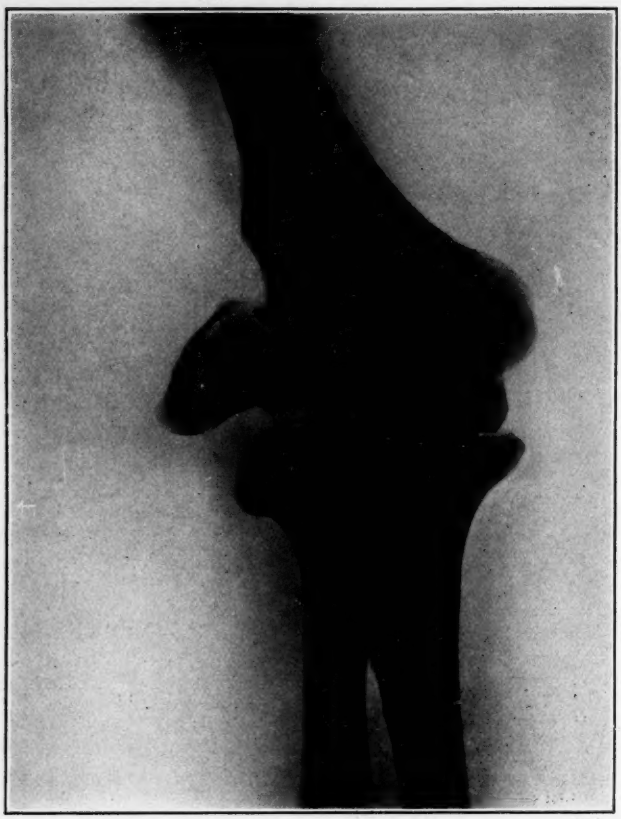

FIG. 272.—*Case* 16. Radiogram of elbow. Late ulnar palsy. Shows: (*a*) The old fracture of the external condyle with non-union; and (*b*) The cubitus valgus deformity.

CONDITION ON FIRST EXAMINATION.—The left elbow presented a conspicuous cubitus valgus deformity, with prominence and apparent over-growth of the internal condylar region. The external condyle showed an irregular projection, and the line of the articular surface of the humerus was oblique. The range of flexion was normal, but extension was restricted to some three-fourths of the full range. The *ulnar nerve* at the elbow seemed to be placed more superficially. It was freely mobile, but did not dislocate. The post-condylar groove was definitely shallow, and the interval between the olecranon and internal condyle appeared to be diminished as compared with the opposite side.

Neurological Syndrome.—

a. Motor function: The ulnar side of the forearm showed distinct atrophy in the region of the muscle bellies of the flexor carpi ulnaris and profundus digitorum, and the ulnar intrinsic muscles of the hand showed a more extreme degree of atrophy. All these muscles were markedly paretic but were still acting. There was no claw deformity.

b. Sensory function: There was a definite hypo-æsthesia to light touch, with an exaggeration of the protopathic response. There were no vasomotor changes or trophic phenomena.

A radiogram (*Fig.* 272) shows a typical fracture of the external condyle, with fibrous union.

OPERATION.—Aug. 15, 1924. Exploration of ulnar nerve; anterior transposition. The operation was carried out with the usual technique. The following special points were noted: (1) The nerve-trunk in the groove was swollen, injected, and

the sheath very thin. There was no palpable induration. Below the level of the œdematous spindle the nerve was wasted and pale. Faradic stimulation above and below the area of the lesion gave a response in the flexor profundus digitorum, but none in the flexor carpi ulnaris or in the ulnar intrinsic muscles. (2) The muscle belly of the flexor carpi ulnaris as displayed at the operation was much attenuated, and consisted practically of a sheet of connective tissue.

SUBSEQUENT COURSE. — Rapid disappearance of the subjective symptoms occurred in the first few weeks after the operation.

Condition Ten Months after the Operation.—The atrophy of the ulnar intrinsic muscles is less marked, and there are signs of increase in power in this group. There is an absence of any sensory loss or vasomotor change. The nerve-trunk is evidently well embedded in front of the elbow, and there is no local tenderness in this region.

Cases 17 *and* 18.—These are two examples of late ulnar-nerve palsy in patients who came under my observation after operative treatment had already been carried out. In both, a considerable amount of bone had been removed from the lower end of the humerus in an attempt to reconstruct a new bony bed for the nerve-trunk. The original bone injury had obviously been a fracture of the external condyle. In each case the result of the operation had been unfortunate, as the clinical signs of a complete nerve-block had developed. Further exploration of the nerve-trunk was therefore considered advisable, and in both cases a large irregular indurated nerve spindle was found embedded in a mass of scar tissue in the region of the old post-condylar groove. The lesions demanded *resection* and end-to-end suture.

These cases are included in the series as they emphasize the dangers of the type of operative treatment which had previously been employed.

CLINICAL FEATURES.

Late ulnar palsy has been seen more frequently in the male sex, as one would expect in a traumatic lesion of this type. It is convenient to recognize three clinical stages which form a definite sequence.

1. **The Fracture.**—The initial injury is sustained in early life, and usually in childhood between the ages of 2 and 10. In the typical case the fracture involves the *external* condyle of the humerus, the line of cleavage running obliquely into the elbow-joint. This is a familiar fracture in childhood, and one which often gives rise to difficulty in treatment owing to the tendency of the large fragment to be 'turned turtle' by the pull of the extensor attachments. It appears to be the rule for this fracture to unite by fibrous tissue only (*Fig.* 273). In many cases the functional result of the fracture as regards joint mobility is fairly satisfactory. The *cubitus valgus* deformity usually manifests itself at a comparatively early stage, but often excites little comment at the time. In adult life the distortion is always conspicuous. This is due in the early stage to the initial displacement of the condyle; later the effects of irregular epiphyseal growth are superadded.

2. **The Latent Period.**—In 75 per cent of the recorded cases the interval between the injury and the onset of the first signs of nerve involvement is not less than ten years. Latent periods of thirty years, or even more, are by no means rare (e.g., *Case* 13). During this time no change in the elbow is noted other than the steady increase in the degree of the deformity, which reaches its maximum with the cessation of growth.

3. **Stage of Neuritis.**—

a. Symptoms.—Signs of interference with the function of the ulnar nerve arise insidiously and usually progress steadily. The neurological syndrome

of the initial phase is found to vary in different individuals. Thus the early subjective symptoms may be predominantly sensory or motor. If the former, the patient experiences local pain in the region of the elbow excited by movements of the joint, together with tingling and a sense of numbness referred to the ulnar area of the hand. When the motor signs take precedence, there is a slowly progressing weakness in the hand, and certain of the finer movements become increasingly difficult to carry out (*Case* 14).

The objective signs at first indicate the presence of an incomplete nerve-block, which as time goes on gradually deepens towards the stage of complete interruption. A partial sensory loss may be present, but it is often overshadowed by the irritative phenomena. The paresis and wasting of the intrinsic muscles of the hand supplied by this nerve soon become manifest. In long-standing and untreated cases a complete ulnar palsy with all its mechanical sequelæ may ultimately be seen. The trophic changes characteristic of an irritative nerve lesion are present in greater or lesser degree, and the patient runs the usual risk of local complications dependent on the existence of an analgesic area of skin.

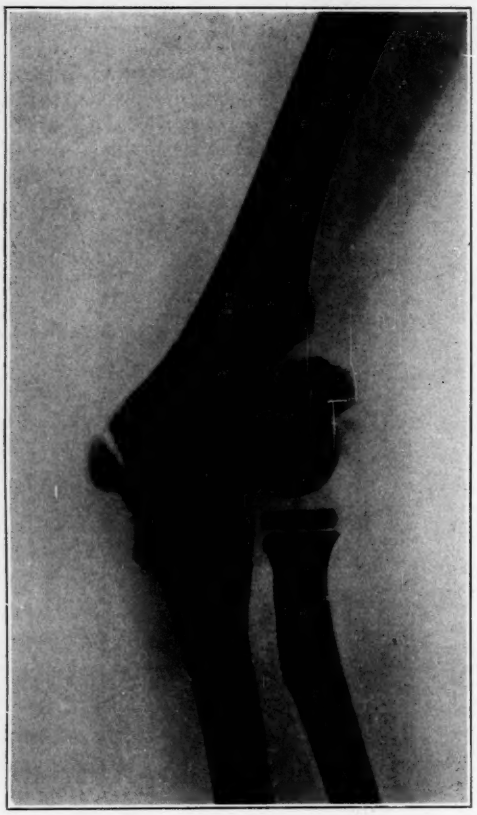

FIG. 273.—Typical fracture of the external condyle in a boy of 12. No bony union seven years after fracture; marked cubitus valgus deformity. No nerve involvement.

b. The Nerve-trunk.—On palpation of the nerve in the post-condylar groove the local lesion is at once demonstrable. At first the nerve is exquisitely tender and slightly thickened; later the local induration is evident in the form of a painful nerve 'spindle'.

c. Configuration of the Elbow.—Reference has already been made to the cubitus valgus deformity which is always a striking feature. The distortion of the lower end of the humerus gives a false impression of overgrowth of the internal condylar region. It is this appearance which has often been responsible for the erroneous clinical diagnosis of old fracture of the *internal condyle.* The ulnar groove is found to be relatively shallow, and in it the tender thickened nerve-trunk appears to lie in an unusually exposed and superficial position.

A radiogram of the elbow demonstrates the old fracture of the *external* condyle, invariably with no sign of complete bony union even at this date (*see Figs.* 270, 271, 272).

PATHOGENESIS OF THE NERVE INJURY.

Mouchet was the first to give a logical explanation of the manner in which the nerve injury is produced. He pointed out that, as a result of the elbow deformity, the nerve is compelled to take a longer course, and tends to be stretched like a bowstring along the inner border of the olecranon. This stretching, however, does not occur during the movement of extension, as suggested originally by Mouchet, for in this position the nerve-trunk is relaxed. On the contrary, the abnormal tension comes into play during each movement of flexion, and the constant repetition of strong flexion movements provides a recurring trauma. It is easy to visualize the gradual development of a 'friction' neuritis under these circumstances, and the lesion illustrates this train of events most beautifully. It is difficult, however, to explain why the onset of the neuritis should be delayed for such a long latent period. From a study of clinical records it appears that the onset of nerve symptoms frequently coincides with a period of strenuous over-activity in which oft-repeated forcible flexion of the elbow is demanded. Such conditions may be provided by the special activities of sport or occupation (*see Cases* 14, 15, 16).

TREATMENT.

There seems to be little scope for conservative non-operative methods in the treatment of delayed ulnar neuritis. Operative exploration has been practised in these cases for many years, but some of the procedures employed have a purely historical interest. Mouchet in his classical article published in 1914 selected four types of operation as being worthy of discussion at that date: (1) *Simple freeing of the nerve* (*neurolysis*); (2) *Gouging out the postcondylar groove*; (3) *Supracondylar osteotomy*; (4) *Anterior transposition of the nerve* (*displacement-neurolysis*).

1. **Simple Freeing.**—This operation was carried out by Potherat in 1897 on Mouchet's first case, but with no amelioration of the neuritic symptoms. In the light of our present knowledge concerning the pathogenesis of the lesion, the operation as a neurolysis is obviously inadequate, and may therefore be dismissed without further comment.

2. **Gouging out the Post-condylar Groove.**—For many years this operation has figured in the literature as a reputable method of dealing with the lesion under consideration. It was practised by Broca in 1899 with disappointing results, but occasional successes have been recorded, e.g., Sherren (1908).

The operation is open to criticism on *a priori* grounds, as it does not fulfil the canons of a properly designed neurolysis. It is clearly unwise to replace a nerve-trunk in a bed where its mobility is likely to be further restricted by the production of a fibro-osseous scar. Moreover, its course is not shortened to an appreciable degree. With the operations at our disposal now to be considered, efforts to enlarge the postcondylar groove cease to be of any practical value. The results of this operation in *Cases* 17 and 18 in my series illustrate its potential dangers.

3. **Supracondylar Osteotomy.**—This is the procedure recommended by Mouchet and practised with excellent results in three cases in his original series. It involves the removal of a wedge, with its base on the inner side, from

the lower end of the humerus, thus correcting the cubitus valgus and at the same time shortening the course of the overstretched nerve-trunk. There is little to criticize in this operation, but it does not seem to have been adopted widely.

4. **Anterior Transposition of the Nerve.**—There are records of this operation having been carried out as long ago as 1898 by an American surgeon, with the report that the pain complained of was relieved. Roux, of Lausanne, operated by this method on one of the early patients seen by Broca and Mouchet, and to him is generally ascribed the introduction of this procedure. In this particular patient, examined some years later by Mouchet, the result was said to be disappointing.

Mouchet's criticism of the operation, as performed at that time, was that the nerve-trunk was left exposed in a subcutaneous position as it passed along its new course in front of the internal condyle. But this technique is no longer followed, for in recent years many opportunities of practising the operation of anterior displacement of the ulnar nerve have been provided in the reconstructive surgery of the late war. As a result of this collateral experience in peripheral nerve surgery, the technique of anterior transposition has been standardized, and the one essential feature is the embedding of the nerve deeply in an intramuscular plane. This operation has gradually established itself as the ideal procedure for the treatment of delayed ulnar neuritis, and has been advocated amongst others by Dean Lewis and Miller, Adson, and Buzzard. It satisfies the primary requirements, viz., to shorten the course of the nerve, and to place it in a bed where both friction and tension are eliminated. In my own hands the operation has proved an unqualified success, and in my judgement is to be regarded as the routine procedure for all cases of late ulnar-nerve palsy.

SUMMARY.

1. Late involvement of the ulnar nerve (delayed ulnar palsy) is a characteristic remote sequela of certain fractures of the *external condyle* sustained in early childhood.

2. *The clinical picture embraces three phases*: (*a*) The fracture in early life; (*b*) The latent period—this is rarely less than ten years; (*c*) The development of neuritis.

3. The neuritis is a friction or tension lesion. It is pre-determined by the existence of a *gross cubitus valgus deformity*. The nerve-trunk is compelled to take a longer course, and becomes stretched like a bowstring in the shallow post-condylar groove.

4. The onset of symptoms is often associated with a period of strenuous use of the limb in which repeated flexion movements of the elbow are carried out.

5. Operative treatment is indicated in all cases. It is necessary to relieve the nerve from friction, and to shorten its course. Two procedures are worthy of adoption, viz.: (*a*) Anterior transposition of the nerve. This is an operation of great precision, easy to perform, and has proved completely effective. (*b*) Supracondylar osteotomy. This is designed to correct the cubitus valgus deformity and so indirectly shorten the course of the nerve. It presents more difficulties than simple anterior transposition.

3. RECURRING DISLOCATION OF THE ULNAR NERVE.

Minor degrees of hypermobility of the ulnar nerve are often discovered in the course of routine examinations of the elbow region, but few individuals develop a true complete dislocation of the nerve from its groove. Of the predisposing causes of hypermobility or luxation little is known. Variations in the depth of the post-condylar groove, congenital or acquired laxity of the nerve sheath or arcuate ligament, and, more especially, an exaggerated 'carrying angle', have all been cited as possible explanations (Stopford[12]). It is true that in a number of the recorded cases an increase in the normal degree of cubitus valgus appears as a definite clinical feature (Schmidt[13]), but it is difficult to explain why this factor *per se* should favour the occurrence of luxation. Although a good many cases have been submitted to operation, the information derived from such a source on this question of etiology has been somewhat indefinite. Thus, in three out of six operations for this disability recorded below (*Series IV*), a genuine laxity of the nerve 'mesentery' could be demonstrated, but the exact significance of this anomaly seems obscure. It is evident that an unusually loose anchorage of this type is present throughout life in a certain proportion of individuals. The first dislocation may then be induced by a single severe trauma. In most cases, however, the luxation develops insidiously and may be unsuspected for a considerable period. The onset of neuritis in a nerve which undergoes habitual luxation may be long delayed, there being a silent period as in the late involvement of this nerve after fracture. The neuritis, when it develops, is a typical friction lesion, with the ultimate formation of a nerve spindle (fusiform neuroma).

SYMPTOMATOLOGY.

It is unnecessary to enter into an extended description of the symptoms of the 'friction' neuritis induced by a slipping ulnar nerve, as the clinical picture is a faithful parallel of the neuritis of the late involvement. Here again, either sensory or motor symptoms may predominate, and the nerve-trunk becomes thickened and tender as the fusiform neuroma slowly appears.

RECORDS OF CASES.

SERIES IV. CASES 19–24.

Case 19.—J. L., male, age 35 (1923).

HISTORY.—Sustained a gunshot wound of the right forearm in September, 1918. Numbness along the inner border of the hand was complained of soon after the injury. A partial lesion of the ulnar nerve was known to be present some years later, but owing to the absence of motor symptoms there was little or no disability. Four years from the date of the wound, signs of an active ulnar neuritis appeared for no apparent reason; the chief symptom was pain and tingling referred to the sensory area of distribution. The symptoms increased steadily in severity.

CONDITION ON EXAMINATION.—There was an inconspicuous wound scar on the mesial aspect of the upper third of the forearm. Full ulnar anæsthesia and analgesia were present, but no signs of paresis or wasting of the intrinsic muscles. There was no tenderness of the nerve-trunk in the region of the wound scar. Examination of

the elbow showed that the nerve was hypermobile and slipped forwards in front of the internal condyle with each movement of flexion. The nerve-trunk in the groove was tender and slightly swollen. There was no exaggeration of the carrying angle.

OPERATIVE TREATMENT was decided upon, and the nerve was exposed (*a*) in the forearm in the region of the old gunshot scar, and (*b*) at the elbow. In the former situation the nerve was lightly adherent, but showed little or no signs of alteration in calibre. It was accordingly left *in situ*, as its surroundings were considered to be suitable. At the elbow the post-condylar groove appeared abnormally shallow; the nerve-trunk was slightly swollen but showed no induration. It was displaced forwards in front of the elbow, and buried according to the usual technique.

Result.—Disappearance of the irritative signs within a few months. The anæsthesia and analgesia have remained unchanged.

Case 20.—**R. W., male, age 30 (1923).**

HISTORY.—Sustained a gunshot wound of the right forearm in 1917 with a compound fracture of the ulna. Healing was slow, and a good deal of stiffness of the fingers remained. There was no evidence of any ulnar-nerve involvement.

Six years later, whilst undergoing a course of training in carpentry, the patient began to feel pain on the inner side of the elbow. This pain was best marked during strenuous efforts at planing and similar manœuvres.

CONDITION ON EXAMINATION.—This revealed a luxable ulnar nerve at the elbow. The nerve-trunk was tender and slightly thickened. There was no increase in the carrying angle.

Conservative treatment was advised and tried for a time; with modified working conditions the pain and paræsthesia diminished. Attempts to resume active work brought on the symptoms once more. Operative treatment was therefore recommended and accepted.

OPERATION.—Anterior displacement of the nerve. In the groove the nerve-trunk was found to be congested, slightly swollen, and just a little indurated. The groove appeared to be unusually shallow, but no other anomaly was recorded.

Result.—Complete relief of the irritative symptoms followed the operation within a few weeks, and there has been no return.

Case 21.—**C. T., female, age 27 (1923).**

HISTORY.—Whilst working in a mill she sustained a contusion of the left elbow. One month later pain was still present and the patient had become aware of the slipping of some structure on the inner side of the elbow during each movement of flexion. Each slip produced local pain and also pain referred to the little finger.

CONDITION ON EXAMINATION.—A slipping ulnar nerve was found; the post-condylar groove was wide and shallow. The nerve-trunk showed neither thickening nor tenderness, and there were no signs of interference with conduction. In view of the patient's occupation, operation was advised.

OPERATION.—Anterior displacement of the nerve. The fascial covering of the nerve in the groove was abnormally lax and resembled a bursal sac.

Result.—Complete disappearance of the irritative symptoms within a few weeks. Patient resumed work and there has been no recurrence of symptoms.

Case 22.—**W. J. L., male, age 40 (1924).**

HISTORY.—Patient complained that for the past six months he had felt something slip on the inner side of the elbow. This occurred during work, and was accompanied each time by a jarring sensation and pain referred along the inner border of the little finger.

CONDITION ON EXAMINATION.—An inspection of the elbow revealed a lax ulnar nerve which slipped forward on to the internal condyle during each movement of flexion. The nerve was tender and somewhat thickened. The faintest trace of

interference with conduction was demonstrated, i.e., hypo-æsthesia in the little and ring fingers, and slight flattening of the hypothenar eminence. There was no increase in the carrying angle. Operative treatment was advised and carried out.

OPERATION.—Anterior displacement of the nerve. The nerve-trunk showed no definite enlargement or induration ; the very thin fascial sheath seemed unduly lax.

Result.—Complete relief of the irritative symptoms.

Case 23.—**J. L. D., male, age 32 (1924).**

HISTORY.—Sustained a gunshot wound of the left forearm in 1918, with an incomplete involvement of the ulnar nerve which was not treated by operation. *Four years later* the nerve at the elbow became painful and tender, and it was found to be hypermobile. Conservative treatment was tried for nearly a year, but the irritative symptoms persisted. Operation was therefore suggested.

OPERATION.—Anterior displacement of the nerve. No striking anatomical anomalies were noted.

Result.—Very marked improvement, but incomplete relief of the irritative symptoms. It was considered that the residual signs were caused by the old gunshot lesion in the forearm.

Case 24.—**W. A. C., male, age 33 (1924).**

HISTORY.—Sustained a contusion of the left forearm which was followed by persistent pain. Pain was referred to the little finger, and the patient was conscious of something slipping.

CONDITION ON EXAMINATION.—A hypermobile nerve was discovered which dislocated with every movement of flexion. The nerve was tender, but there were no signs of interference with conduction. It was doubtful whether the symptoms were associated entirely with the slipping nerve ; but as the patient had become increasingly aware of the recurrent luxation, it was thought wise to operate. There was no increase in the carrying angle.

OPERATION.—Anterior displacement of the nerve. The nerve appeared to be slightly flattened, and the hypermobility was due to an unduly lax covering sheath ; this was almost mesenteric in its attachment.

Result.—Complete abolition of the irritative symptoms in the elbow region.

TREATMENT.

1. **Recurring Dislocation without Neuritis.**—In cases where the luxation has been recently discovered and before the onset of signs of definite neuritis, it is advisable to give a trial to conservative measures unless there are strong economic reasons for urging early operation. If it is possible to enjoin a limited use of the elbow with the avoidance of repeated forcible movements of flexion, the stage of neuritis may be postponed almost indefinitely. Operative treatment should be considered, however, in individuals who are engaged in manual occupations, or in others who are unable to submit to the necessary restrictions demanded by conservative treatment.

2. **Recurring Dislocation with Neuritis.**—With the appearance of the first symptoms of neuritis, conservative treatment has little to offer ; in such cases operation should not be delayed. Various elaborate procedures have been employed in the past ; but there can be little doubt that there is one type of operation only which is applicable for this lesion, viz., *anterior transposition* of the nerve-trunk. The objections to the older or alternative operations have already been fully stressed. As in other forms of traumatic neuritis of this nerve in the post-condylar groove, the operation of anterior displacement is followed by results which are exceedingly gratifying.

THE OPERATION OF ANTERIOR TRANSPOSITION OF THE ULNAR

A full description of this operation was given by Stiles[14] some years ago, and more recently by the writer in Carson's *Modern Operative Surgery*.[15] It seems worth while to consider here quite briefly the main technical features of an operation which has proved to be so effective in the treatment of several types of traumatic ulnar neuritis, and also has a much wider application. I have now employed this procedure on some 90 occasions.

As already mentioned, the operation came into prominence during the hey-day of the peripheral nerve surgery of the war. The indications for its employment may be summarized as follows :—

1. As an aid to *end-to-end* suture in gross lesions of the nerve with extensive loss of continuity. Thus a series may be quoted of 115 ulnar-nerve sutures for gunshot lesions performed by the writer, in 55 of which it was necessary to displace the nerve in front of the elbow.[16]

2. As a form of *neurolysis* for all incomplete lesions of the nerve in the post-condylar groove : (*a*) Gunshot wounds and other severe traumata ; (*b*) The various types of ulnar neuritis described in this paper. It may be stated here, as a fundamental principle, that when it is necessary to explore at operation an incomplete lesion of the ulnar nerve in the post-condylar groove, the nerve-trunk should never be returned to its orginal bed. In all such cases this bed is now unsuitable, and the nerve should be displaced forwards and buried deeply where it is no longer exposed to abnormal tension or friction. This is the ideal neurolysis.

The operation of anterior displacement comprises the following steps : (1) Generous freeing of the nerve-trunk in the lower third of the upper arm, in the groove, and in the upper third of the forearm. In the latter situation access is gained by splitting the line of fusion of the two heads of origin of the flexor carpi ulnaris. (2) The proximal branches of the nerve must be carefully freed to allow the nerve-trunk to be fully mobilized. It is necessary to sacrifice the fine branch to the elbow-joint, but great care must be taken to conserve the branches to the flexor carpi ulnaris and flexor profundus digitorum which arise in the lower part of the groove. These twigs are stripped up in a proximal direction from within the sheath of the parent trunk, and also stripped distally to increase their extramuscular course. In this way kinking is avoided, and the ulnar trunk can be easily displaced well in front of the condyle. (3) The new nerve-bed must be situated deeply in an intramuscular plane. It is constructed either (*a*) by division of the superficial fibres of the forearm flexors taking origin from the internal condyle, or (*b*) by the detachment and turning down from the condyle itself a tongue-shaped muscular flap (Bristow). (4) Where the nerve-trunk passes forwards in the upper arm across the line of the intermuscular septum, this structure must be exsected over a sufficient area. Unless this is done, the nerve is likely to be bow-strung over a tense fascial bridge.

SUMMARY.

1. There are certain special types of ulnar-nerve injury which are primarily determined by an alteration in the normal relation between the

nerve-trunk and its bed in the postcondylar groove. Under such conditions the nerve becomes exposed to the cumulative trauma of repeated overstretching or friction.

2. The nerve lesions produced in this way are ordinarily of the incomplete type, and may be described under the broad title of traumatic neuritis.

3. Three clinical groups may be distinguished: (*a*) Ulnar-nerve injuries associated with recent fractures of the lower end of the humerus. In these, spontaneous recovery is the rule; operation is called for in rare cases of severe and persistent neuritis. (*b*) Late ulnar-nerve involvement after fractures. (*c*) Recurring dislocation of the ulnar nerve. In the two latter groups operative treatment is indicated as a routine.

4. There is one simple and effective operation which is universally applicable in all cases of traumatic neuritis of the ulnar nerve in the postcondylar groove, viz., *anterior displacement* of the nerve-trunk.

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OPERATIVE REPAIR OF CRUCIATE LIGAMENTS IN SEVERE TRAUMA OF KNEE.

By ALEXANDER H. EDWARDS, Glasgow.

OPERATION.

An incision is made commencing over the centre of the lateral condyle of the femur. It passes at first distally and parallel to the long axis of the limb, but soon curves dorsally, keeping about one inch lateral to the anterior tubercle of the tibia and the same distance beyond this landmark. From the latter point it curves in a proximal direction behind the medial condyle of the femur. At the level of the centre of this condyle it passes proximally parallel to the axis of the limb, and ends at a point or level 3 or 4 in. from the proximal margin of the patella. The incision is roughly of horseshoe shape. The skin and subcutaneous tissues are to be reflected to the fullest extent of the incision, after which the sartorius muscle is clearly defined, and drawn aside to allow free access to the gracilis and semitendinosus tendons (*Fig.* 274). These should be dissected with their sheaths intact. After they have been exposed in their entire length, they are divided close to their insertions (*Fig.* 275). The ends are then grasped in forceps, covered with protective gauze, and laid aside till a later stage.

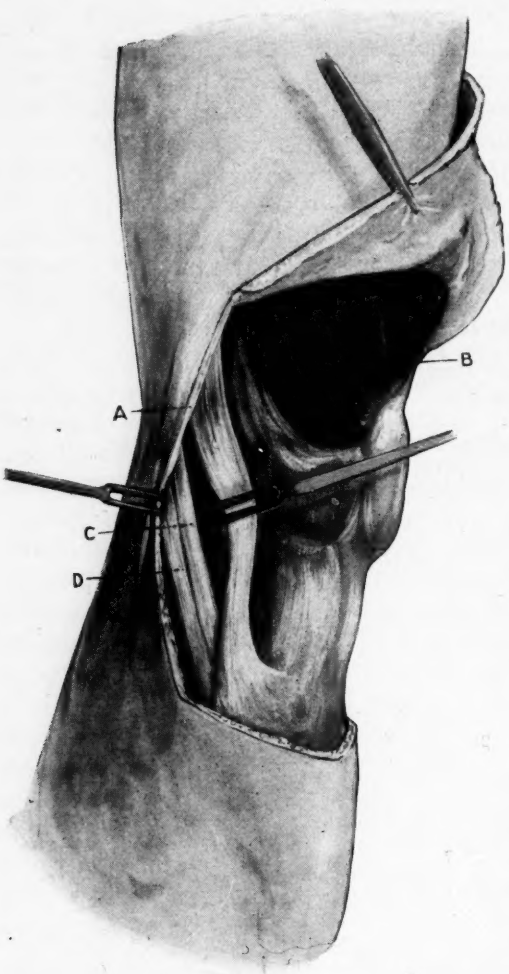

Fig. 274.—Showing skin and subcutaneous tissues reflected to expose tendons. A, Sartorius; B, Vastus internus; C, Gracilis; D, Semitendinosus.

The next step is to divide the deep fascia and attachments of the vastus medialis to the proximal (upper) surface of the medial

condyle. The bone at this point is to be cleared with a detacher, so as to allow drilling of a passage from the newly cleared area through to the centre of the joint cavity. The area covered by the point of the drill is immediately ventral to the adductor magnus tendon. Next the deep fascia close to the medial tuberosity of the tibia is exposed, and the bone here laid bare, slightly dorsal to an imaginary line passing through the centre of the medial femoral condyle and parallel to the long axis of the limb. On the lateral aspect of the tibia the corresponding area of bone is exposed.

A free exposure of the joint cavity is now made through an incision, horseshoe shaped, on the lines of the first incision, though of less extent. The patellar tendon is thrown up with an osteoplastic flap from the tibial tubercle (*Fig.* 276). In exposing the joint, the greatest care should be taken to avoid cutting either of the lateral ligaments. These should be examined for signs of laceration, and if possible repaired after the cruciate ligaments have been dealt with.

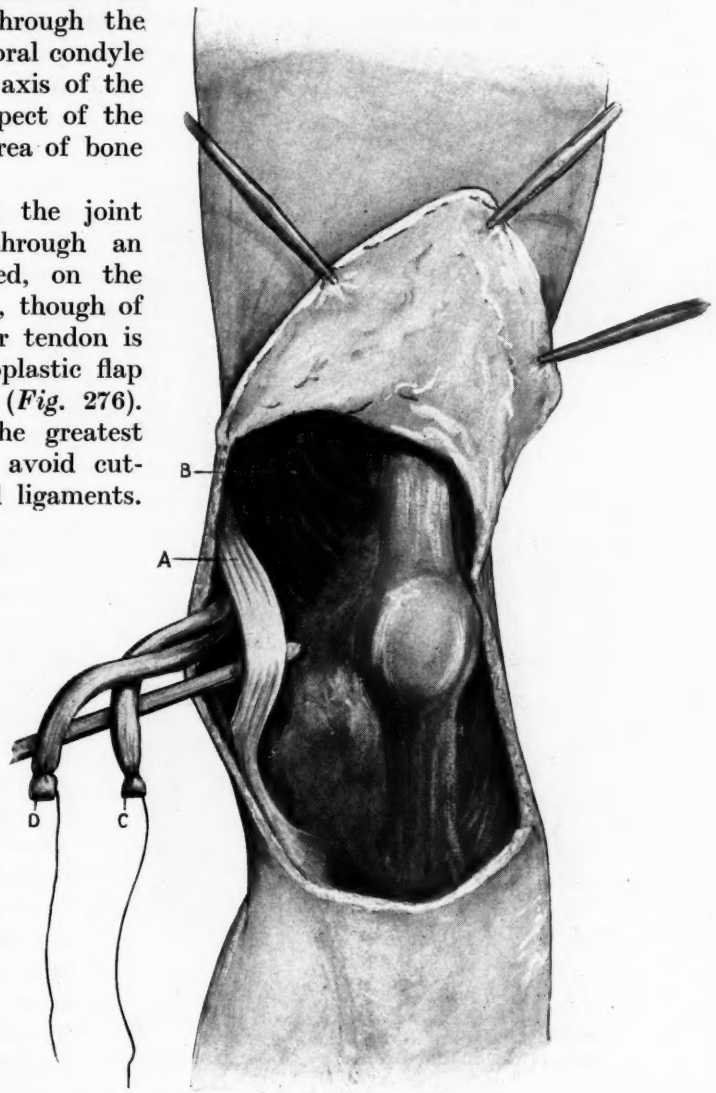

FIG. 275.—Showing tendons divided. A, Sartorius; B, Vastus internus; C, Gracilis; D, Semitendinosus.

A drill, $\frac{1}{4}$ to $\frac{3}{8}$ in. diameter, 4 to 5 in. long, for the tibia, and $\frac{3}{8}$ to $\frac{1}{2}$ in. diameter for the femur, should now be requisitioned to make the new bone canals necessary in the femur and tibia. The tibia should be drilled on each side, the point of the drill being applied to the areas already prepared, and the lines converging in a proximal direction to meet in a common opening at the tibial spines (*Fig.* 277). The drilling will probably be best done by hand, as the drill is thus more easily controlled, and with ordinary care the proper working angles can be maintained. It will be necessary to have the knee slightly flexed to complete this part of

the operation, but for the drilling of the medial condyle the flexion must be full. The new canal begins at the part close to the adductor magnus tendon already mentioned. As a guide to the angle, a long thick probe may be passed through the opening in the lateral tuberosity of the tibia, and the line to follow is that towards the centre of the intercondylar space, or slightly dorsal to this. All bone débris should now be cleared from the canals made and from the joint cavity; the tendons are then uncovered, and long silk sutures fixed securely through their distal ends. Long forceps are pushed through the femoral opening from the joint side, and by means of them and the silk ligatures the tendons are pulled into the joint. The tendons are now crossed, or twisted (*Fig.* 278), the semitendinosus being drawn through the lateral tuberosity of the tibia, and the gracilis passed through the other artificial canal (*Fig.* 279). They are now pulled as tight as possible with the knee fully extended, after which, the tension being still maintained, they are fixed down securely with silk to the deep fascia of their respective areas. The osteoplastic flap is returned to its place and nailed down, the joint capsule being carefully sutured thereafter (*Fig.* 280). After closure of the superficial wound, the knee should be fixed on a splint in slight flexion till healing is complete, and as soon as possible thereafter a walking plaster applied to include the foot, knee, and pelvis.

FIG. 276.—Exposure of knee-joint by raising patellar tendon with an osteoplastic flap. C, Gracilis; D, Semitendinosus.

The operation just described was examined on the cadaver by Professors Bryce (Glasgow) and Waterston (St. Andrews) in their respective Anatomy Departments. Both of these eminent authorities expressed approval of it as an anatomical proposition, whilst Mr. J. Hogarth Pringle, F.R.C.S., lately surgeon to the Royal Infirmary, Glasgow, expressed equal approval from the surgical standpoint.

REMARKS.

The above operation was carried out experimentally in the Anatomy Department of the University of St. Andrews in 1918, with the very kind permission of Professor Waterston. No opportunity for its practical application has occurred since that year. It is published in the hope that in some cases it may be found useful. In the course of some years of work in examining pensioners, the writer has seen a number of cases of flail knee. Throughout the United Kingdom, and in all countries involved in the Great War, there must be a large total number of patients suffering from this disabling condition. The operation of ankylosis of the knee has been frequently done, and this classical procedure may have been for the surgeon the simplest way out of the difficulty; but it may not have been the ideal solution, for even a perfect ankylosis still precludes the possibility of the patient following certain common occupations, thereby diminishing the wage-earning average.

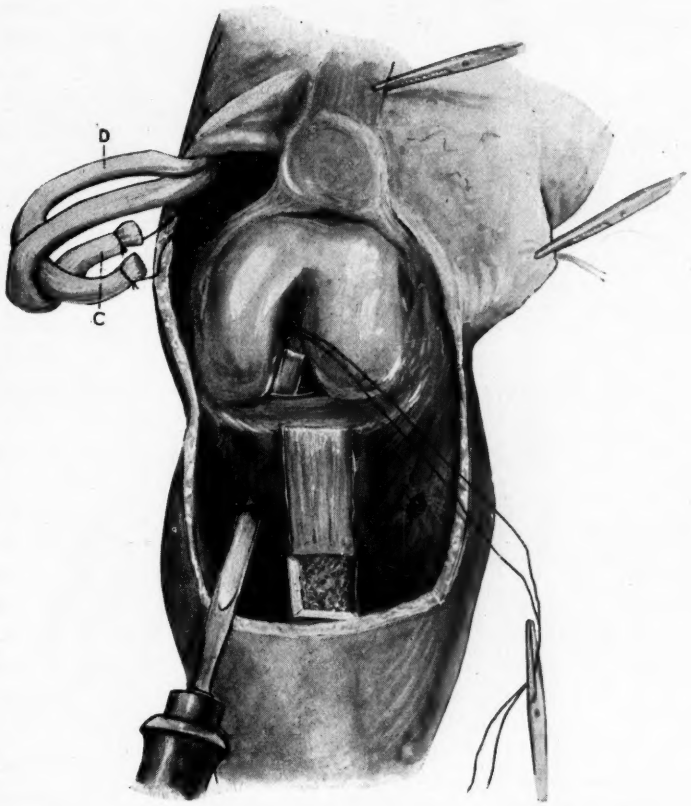

FIG. 277.—Showing the holes drilled in the bones through which the tendons are passed. C, Gracilis; D, Semitendinosus.

The object of this paper is not to discuss primary treatment, operative or conservative, but to attempt to offer a scheme of constructive surgery which may be of service in cases of flail knee resulting from destruction of the cruciate ligaments. In such cases, where conservative treatment has failed, it is hoped that surgical interference on these lines may be considered. Jones and Lovett, in their text-book of orthopædic surgery, state that they have examined several cases of "old flail knees with marked functional defect", and after reconstructive operation they had not seen a perfect result, though several had been much improved.

Various reconstructive methods have been devised. In one, the iliotibial band is transplanted, and an attempt made to convert it into an intra-articular ligament (Aylwin Smith). It is thought that a serious objection

to the use of this structure as a graft is that it is practically avascular, and in the operation referred to it is separated from its source of vascularity. Post-operative rest, added to loss of direct nutrient blood-supply, must lead to its degeneration and subsequent yielding, as it is a comparatively low type of tissue histologically. In another operation recommended (Hey Groves), the semitendinosus and gracilis muscles are isolated close to their muscle bellies, the latter being anchored to the semimembranosus. The two tendons are then put through a transverse drill-hole in the medial condyle, and ultimately fixed to the head of the tibia. If this operation does not give perfect results, this might arise: (1) From the circuitous route pursued by the tendon grafts; or (2) Principally because the tendons are separated from their corresponding muscles, whereby there is interference with their nutrition. The writer ventures to think that it has not yet been demonstrated that synovial fluid alone will adequately nourish tendon grafts. In primary suture of torn cruciate ligaments, success is due to the fact that their basal blood-supply (which proportionately to their bulk is considerable) has not been destroyed; after a certain period has elapsed simple suture probably will be futile—as in all likelihood is the case also with the method of using grafts of iliotibial band at any stage.

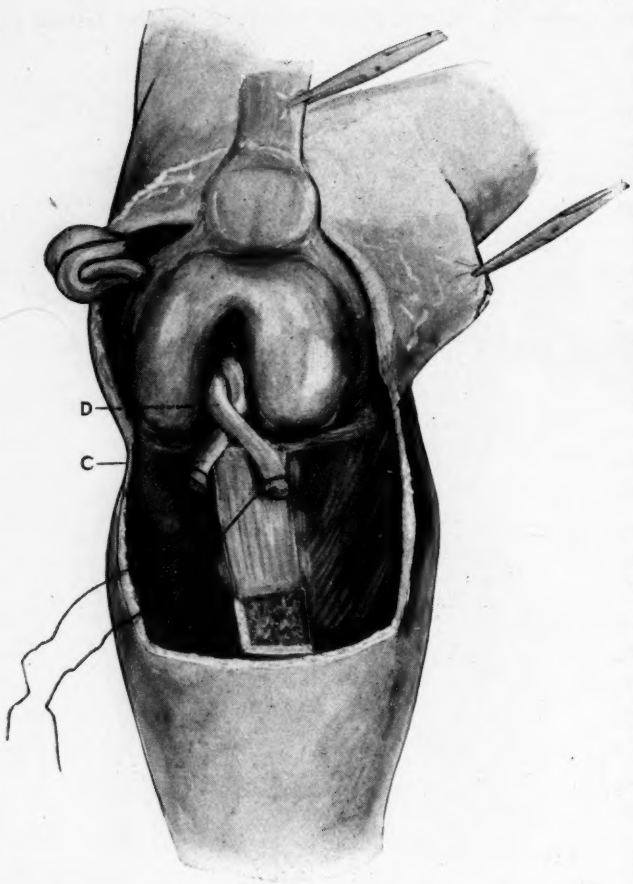

FIG. 278.—The twisted tendons are drawn through the canal in the femur. C, Gracilis; D, Semitendinosus.

An important effect of rupture of one cruciate ligament is that it throws exceptional strain on the other, and also on the collateral ligaments. In course of time, as abundant clinical observations have shown, anteroposterior movement of the tibia on the femur develops. As these ligaments control medial rotation of the tibia, rupture of one must result in undue tension on the other when this movement occurs. If both ligaments are torn, the strain on the collaterals must be still more greatly increased in rotation. But what happens as a modification is that the condition is treated, as it often has been, by expectant methods for a very prolonged period, and that

extreme atrophy of bone, cartilage, muscles, and ligaments results. So that to the looseness due to a torn cruciate is added the laxity of weak flabby stretching structures in and around the joint.

In the *Annals of Surgery*, August, 1907, Mr. J. Hogarth Pringle described a case of ruptured anterior cruciate ligament with avulsion of its tibial attachment. Twelve days after reception of the trauma, the lesion was treated successfully by suture, and the ultimate functional result was very good. In a second case, this surgeon found the anterior ligament torn from its condylar attachment. The operation for suture was done about nineteen months after the primary accident, and the result was satisfactory. The first case exhibited no anteroposterior movement of the tibia on the femur; but the second did show this at the time of the operation, though not at the time of the primary injury.

A very important sign in both these cases was abnormal abduction of the leg with the knee fully extended, and Mr. Pringle formed the opinion after experiments on the cadaver that this was caused to some extent by rupture of the deep fibres of the tibial collateral ligament. It is obvious, at all events, from the clinical signs reported by this surgeon, that instability of the joint was well marked and most probably would progress in the absence of successful operative treatment.

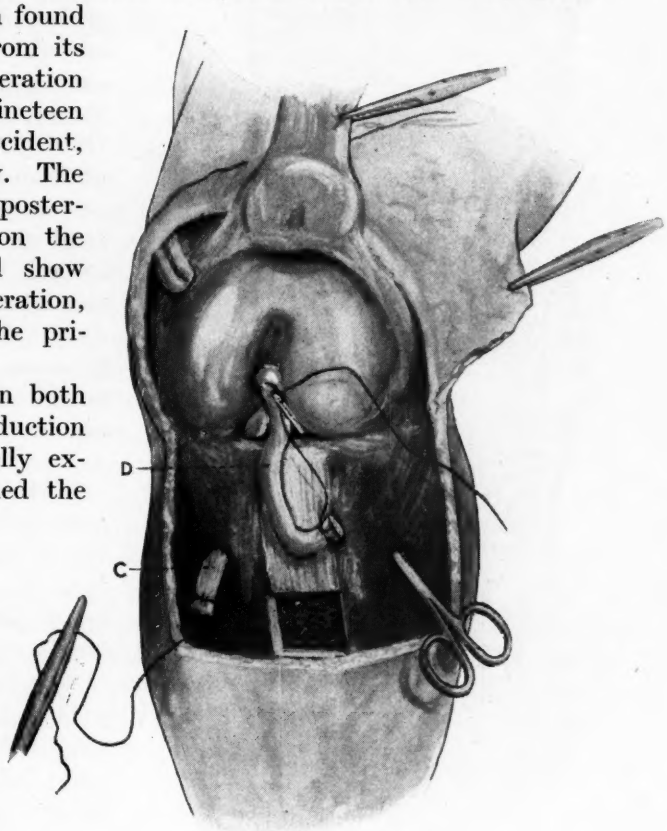

FIG. 279.—Showing the semitendinosus (D) being drawn through the lateral tuberosity of the tibia. The gracilis (C) is already in position.

The main points which have led to the publication of this plan of operation are:—

1. The lines of stress of the normal ligaments have been imitated as far as possible, and with this end in view the transplanted tendons have been crossed in the joint. This arrangement, it is thought, will assist to prevent hyperextension.

2. The new ligaments remain attached to their muscle bellies for maintenance of nutrition.

3. In the cadaver hyperextension was impossible under degrees of strain much higher than would occur from ordinary use in the living.

4. The muscles and tendons, being continuous, would probably in the

living subject maintain a ligamentous activity, which is of great physiological importance.

5. Flexion to a right angle was easily accomplished in the cadaver, and probably a wider range would be possible in the living.

6. Rotation of the tibia is controlled by the crossing or twisting of the transplanted tendons. This arrangement would tend to reduce strain on the collateral ligaments, thereby assisting in the maintenance of stability.

7. The semitendinosus is closely applied to the medial aspect of the lateral condyle and the gracilis to the lateral surface of the medial condyle. This emplacement of these tendons in all probability would counteract lateral movement, and also aid in controlling rotation.

FIG. 280.—Showing the osteoplastic flap replaced and fixed and the tendons sutured.

It has been stated by high authorities that such extensive operations on the knee are so serious that they should not be attempted. In reply to this it might be said that almost everything depends on the technical skill of the operator in avoiding sepsis. The knee-joint is operated on daily for loose cartilage, and with good results, and brain surgery has been for a long time on a definite basis. With a scrupulous aseptic technique there should be no more dread of sepsis here than in any other field of surgery. It is recommended that the operation suggested should be first practised on the dead subject.

TUBERCULOSIS OF GLANDS IN THE ILEOCÆCAL ANGLE: A CAUSE OF PAIN IN THE RIGHT ILIAC FOSSA.

By L. R. BRAITHWAITE, Leeds.

It is not to be assumed that the writer has any new facts or figures to bring forward on a subject about which such a vast literature exists; the failure to diagnose a condition which is fairly common is the excuse for publishing this article.

In order that the subject may be confined within reasonable limits, tuberculosis of the cæcum, small intestine, or mesentery is excluded, and the author confines himself as far as possible to tuberculosis of the glands which lie roughly in the ileocæcal angle and where no demonstrable primary source of infection exists. Those numerous complications which may occur, and which give rise, by bands, kinks, and adhesions of the intestine, to intestinal obstruction, are also excluded. The subject is to be considered (as regards its diagnosis and its treatment) almost wholly in relationship to appendicitis, and especially to the subacute and chronic varieties.

It is probably correct to say that, as ovariotomy, certain operations done for intestinal stasis, and gastro-enterostomy for the atonic stomach have passed into history, operations done for so-called *chronic* appendicitis are more unsatisfactory in their results than almost any others.

There is no doubt about the frequency of chronic pain in the right iliac fossa, and the author hopes to show that some of these cases are to be considered as potential cases of tuberculosis of the ileocæcal glands.

It will be readily understood that whilst in young children the disease may be in the active stage, in adults the causative factor of the pain is usually the remains of a long dead infection.

One great reason for bringing the subject forward at this time is the rapid advance in X-ray diagnosis, the author being fortunate in having the assistance of one of the most brilliant of its exponents.

FREQUENCY.

There is much truth in the dictum that traces of old tuberculous mischief can be found in the chest and in the abdomen of nearly all of us.

It is probably true that old tuberculous disease of abdominal lymphatic glands is very commonly found in the process of operations done for other abdominal conditions. Most of these give rise to no symptoms, and by some surgeons are not regarded with more than a passing interest. It is a very difficult thing to obtain figures to illustrate the incidence of the disease, because only rarely has any note been made bearing on it. It follows that where a particular surgeon habitually *looks for* and notes the presence of diseased glands, there is a very high incidence in his cases.

At the Leeds Infirmary up to 1912 the presence of these glands in abdomens opened for operations of various kinds was practically unrecorded. As a result of a request by the author, the numbers noted began to increase year by year, until in 1924 77 were noted.

Out of 58,731 in-patient cases of all kinds operated upon at Leeds General Infirmary between 1912 and 1924 (inclusive), only 435 were stated to have tuberculous abdominal glands, made up as follows :—

	Males	Females	Total
Cases operated on ..	35,167	23,564	58,731
Number with tuberculous mesenteric glands ..	228	207	435

These are made up as follows :—

Ages	Males	Females	Total
Up to and including 10 years ..	46	38	84
Over 10 but not over 20 ..	80	67	147
„ 20 „ „ 30 ..	49	47	96
„ 30 „ „ 40 ..	26	32	58
„ 40 years of age ..	27	18	45
No age given	—	5	5
	228	207	435

As showing the effect of a personal interest, more than one-third of these cases were under the care of the author of this paper.

The difficulty in arriving at a true estimate is again shown in an extraordinary diversity in the figures in different areas. As is well known, surgical tuberculosis is found more often in certain areas in Scotland than elsewhere. It is not surprising to find that the disease is rife in a very limited area and apparently absent in areas contiguous to this. In the author's experience a series of cases has twice been traced to one 'milk round'; in both infected cows were found to be the source of the trouble.

It is remarkable to find that the percentage of tuberculous abdominal gland cases is higher in private than it is in infirmary practice. The author has himself operated upon over one hundred cases in private practice, and about an equal number in the very much bigger infirmary practice.

Corner believes that tuberculous mesenteric glands are to be found in practically every child in which an abdominal operation is necessary.

Risley believes the disease to be extremely common in infancy and childhood but by no means confined to this period, being nearly as common in young adults. In fact, the highest percentage favours the years from 16 to 18.

Eisenhardt, in 1000 post-mortems of adult phthisis cases, found that 563 had intestinal tuberculosis; Ronseff in 800 found 46 per cent; and Harnan, in 832, 68 per cent.

Struthers says in the years 1919–20 he met with 22 cases of mesenteric adenitis, whilst he dealt with 187 cases of appendicitis; most of the cases were young children.

Shiota, reporting 24 cases from Japan, makes the interesting observation that *none* of them had drunk milk.

Franke in 1914, at the forty-third Surgical Congress in Berlin, reported 90 cases in which operation measures were taken.

Maclaren in 1916 reported 100 of these cases during four or five years, some of them sent for the purpose of having their appendices out.

Frankel, of the North Chicago Hospital, writing in 1917, says: "All statistics show the rarity of intestinal tuberculosis in the first year of life, the frequency of the disease gradually increasing until the fourth or fifth years, and then diminishing again. This term coincides with the average time of weaning and change from mother's milk to the diet through which contaminated food has an opportunity to invade the intestinal canal".

It is interesting to find that according to Scott, of Washington, only 5 per cent of all appendices removed are tuberculous; whilst according to Lockwood there are 2 per cent, and according to Tetz the figure is 3·11 per cent.

Dr. Dingwall Fordyce, writing of the Royal Hospital for Sick Children, Edinburgh (1909), says the disease is extremely common—in the Edinburgh district at least.

Goodhart and Still say that although 59 per cent of all tuberculous post-mortems showed tuberculosis of mesenteric glands, only 46 cases were diagnosed clinically in 6000 to 7000 patients—half of them being very doubtful.

Carson gives the age incidence as follows:—

	Cases		Cases
Between 1 and 5 ..	8	Between 15 and 20 ..	2
„ 5 „ 10 ..	14	„ 20 „ 25 ..	4
„ 10 „ 15 ..	15	„ 25 „ 30 ..	5
		Over 30	2

Of the 50 cases, 29 were males and 21 females.

Corner (1905) says mesenteric glands are the most frequent source of tuberculous infection of the body. In 1912 he writes that in children with abdominal signs there are cent per cent of tuberculous abdominal glands. He quotes Professor E. Sims Woodhead as finding 78·7 per cent in autopsies on tubercular children.

Dr. Sabourin, of Durtol, in 1913, makes the very interesting observation in respect of sanatorium patients that in each batch of 50 boarders 20 to 25 suffer in respect of their 'appendix'—i.e., symptoms in the right iliac fossa.

Opie, of the Washington University (1920–1921), says few instances of healed tuberculosis of the mesenteric lymph nodes are found among children or adults examined in the city of St. Louis. In a series of 93 autopsies made in 1917 upon children, and of 50 autopsies made on adults, no instance of healed mesenteric tuberculosis was found. Nevertheless it is probable that primary tuberculosis of the gastro-intestinal tract occurs in a small proportion of children in St. Louis, as elsewhere. An impressive contrast has been furnished by autopsies performed on British soldiers at Base Hospital 21, stationed at Rouen, France, for they have demonstrated the surprising frequency of healed calcified, occasionally still partially caseous, or even frankly caseous, lesions of the mesenteric lymph nodes. Healed focal and lymphatic tuberculosis of the lungs similar to that seen in St. Louis was common, and often the lesions were very extensive; but instances in which little of any old pulmonary tuberculosis was demonstrable appeared to be

much more common among the British than in St. Louis, where the former observations had been made. It is not improbable that the lower incidence of the lesions of the lungs bears some relation to the greater frequency of localized infection by way of the intestinal tract.

Caseous or calcified mesenteric nodules were found in 18 instances among 66 young male adults, whose ages, with few exceptions, varied from twenty to thirty years. Among 15 autopsies, of which notes were lost, the proportion of mesenteric tuberculosis was approximately the same. In a number of instances X-ray plates were made from both the mesentery and the lungs, and they serve to show with considerable accuracy the size and position of the calcified lesions which were present.

SYMPTOMATOLOGY.

It is convenient to consider at this point whether a non-tuberculous adenitis of the ileocæcal glands is a condition likely to occur and to give rise to symptoms.

There is no doubt that in animals, especially cats, simple ileocæcal adenitis is exceedingly common; in man a much greater immunity from adenitis is likely, but it is fair to suggest that the condition may occur.

In the BRITISH JOURNAL OF SURGERY (July, 1923) the author writes of ileocæcal adenitis and its possible bearing on the causation of duodenal and gastric ulcer. Is it not likely that some of the pain so commonly complained of in the right iliac fossa may be due to a simple inflammatory condition of these glands, infected from the contiguous bowel?

The symptomatology of tuberculous ileocæcal glands divides itself into two definite groups: (1) *In children*; (2) *In adults*.

The first group comprises two sections (*a*) *acute*, (*b*) *subacute*, which depend upon the degree of acuity of the adenitis. The second group is to be considered as the chronic stage with subacute exacerbations. It will be clear that no definite line can be drawn between these varieties, because the acute is sometimes, though very rarely, met with in adults, while the chronic is sometimes seen in children.

Speaking broadly, *Group* 1 consists of cases with an acute or subacute adenitis, whilst *Group* 2 includes only cases in which, natural repair or resistance having been achieved, the resulting products, i.e., caseation and calcification, are responsible for the symptomatology and physical signs. It follows, therefore, that *Group* 1 is not commonly capable of a diagnosis by X rays, whereas *Group* 2 usually is.

Group 1.—**Children from 2 to 10 Years.**—It is a fallacy to suppose the child bears the usual stigmata of tuberculosis. In cases where the tuberculosis diathesis is typical, i.e., in children of poor parents and living in unhygienic conditions, it is much more common to find the disease involving not only the glands but the mesentery, the omentum, and the last part of the ileum. The typical case of tuberculosis of ileocæcal glands is one where the child is fat, phlegmatic, and pallid, usually of good parentage, and living in perfectly healthy surroundings. As a rule there has been no history suggesting tuberculosis anywhere; occasionally, however, one hears of cervical glands which have given rise to anxiety.

a. Acute—In children from 2 *to* 6 *years.*—The onset can only be compared with that of an acute appendicitis. It is sudden and violent; the child draws up its legs, the abdomen is rigid, and tenderness, although present all over it, is chiefly recognized in the right iliac fossa. The pulse rises rapidly to 120, the temperature to 100° or even to 103°; respirations are increased in number, but more shallow in excursion. Vomiting ensues and the tongue becomes furred. Is it possible for anyone under these circumstances to take the risk of putting aside the possibility of acute appendicitis?

It has been suggested that an acutely inflamed gland causes a rise of temperature, whereas acute appendicitis does not. How many men in general practice will agree with this? It is known that almost every acute painful condition in a child gives rise to an elevation of temperature. A temperature above 100° would indicate the condition to be tuberculous adenitis rather than appendicitis.

Painful micturition, especially pain at the end of the act, is certainly to be found in most cases of acute appendicitis; but is it not likely, in order to have this symptom well marked, that the appendix must lie in close contact with the bladder?

Operation should therefore be undertaken for acute appendicitis with a reservation that the condition may be an acute adenitis; sometimes both conditions are present.

If there is present a serious degree of pain with a rigid abdomen, tender chiefly in the right iliac fossa, and if this condition lasts for twelve hours, operation should be undertaken on the assumption that acute appendicitis *may* be found. It is perfectly well realized that acute attacks of pain in children are common enough. Many are cured by a dose of castor oil; a few are lost; it is for the few, maybe the very, very few, that one pleads.

Assuming that operation is undertaken, what is found when the appendix is not apparently the cause of the trouble? Unless the ileocæcal and the mesenteric glands which drain the last six inches of the small intestine are examined, nothing will be found, and the case will be considered one of appendicitis without any obvious change in the organ or one of 'thrombosis of the appendical vessels', or a 'stenosis' will be found in its lumen. There is no doubt in many cases an acute adenitis is present, and is shown by enlargement and redness of this group of glands. Sometimes it is shown by a red areola invading the mesentery along the circumference of one or more.

One may quite well imagine a condition of this kind to result from a simple intestinal toxæmia; but the association of exactly similar changes in the same group of glands with areas of caseation and calcification makes it clear that many at least are tuberculous. It is not suggested that anything more than appendicectomy should be attempted. Probably in a very few cases this is the primary focus; but it is more likely by far that the condition arises as the result of infection in the last few inches of the small intestine, and the starvation and general treatment which follow an abdominal operation cure the acute condition.

b. Subacute—Usually in children from 6 *to* 10 *years.* Similar in type to those in (*a*). In this condition we are dealing probably with the repair of a gland, and not, as in (*a*), with the onset of the disease. The symptoms are

somewhat similar but less marked: repeated attacks of sudden abdominal pain, suggestive of intestinal colic (in fact they may be attacks of colic).

With the onset of pain the child cries out, holds its belly with both hands, draws up its legs, and in ten minutes is perfectly fit and well. Occasionally there is vomiting; more rarely there is a passing rise of temperature to 100°. The child, though appearing in pain during the attack, is perfectly well immediately after and before. Deep palpation may reveal a tender spot in the region of the appendix. There may be two or three attacks in a day, or there may be intervals of months between each attack.

Again the question of appendicitis arises! Can anyone in our present state of knowledge be certain that this is *not* appendicitis? It is true that in this group of cases one has more time to think, and operation is not so clearly indicated as in (*a*); but unless the child is under close observation the assumption of tuberculous adenitis is a risky one.

As a rule these cases are diagnosed as 'appendicular colic' or possibly 'subacute appendicitis', and operation is usually undertaken deliberately in an interval. A perfectly normal appendix is found and removed; the absence of any inflammation is accounted for by the fact that only an 'appendicular colic' is being dealt with, or a 'concretion in the lumen' is held to be sufficient cause. Unless examination is made of the glands in the ileocæcal angle and in the mesentery of the last six or eight inches of the small intestine, the real cause of the pain will remain undiscovered, and recurrence of the attacks will be certain. Removal of the affected gland will ensure a cure of the condition; but even if it is not removable, or if the condition is widespread and therefore probably inoperable, one will be in a position to explain to the parents that recurrence of pain may occur, and one will also be able to assure them that, although the child may have pain, the origin of the pain is known, and if the appendix is removed there is nothing left which could give rise to a fatal issue.

It is, of course, perfectly recognized that, assuming the condition is left alone, a natural cure will eventually take place.

Diagnosis in this class may quite well be made by an X-ray examination; and assuming this to be the position, there would be some ground for holding one's hand as regards operation, though there would be considerable risk to be taken in doing so, and considerable anxiety to everybody concerned.

Between the ages of 10 and 15 years there appears to be a period when the disease is not commonly evident.

Group 2.—**Adults from 15 to 45 Years.**—This type represents the common case of *chronic* aching pain in the right iliac fossa; it is slightly more common in women than in men. The patients as a rule are thin and anæmic, usually constipated and of a 'nervous temperament'; sometimes they are apparently perfectly well, fat and ruddy, and certainly not nervous.

The complaint of pain is over a period of years, with long intervals of freedom. The site of the pain is about MacBurney's point, sometimes above it, but rarely below; it is a dull ache, a drag, or occasionally a stab. When the gland is pressing deeply into the iliac fossa there is sometimes pain

suggestive of renal disease, and sometimes pain increased by full extension of the leg at the hip, either on walking or playing active games, such as tennis. Relief is sometimes achieved by lying down, though in a bad attack this may be by no means the case. There are no constitutional disturbances, and there is no rise of temperature.

It would appear that the cause of the pain is pure irritation, a peritonitis at the surface of one or more glands, due to the presence of caseous material, or to actual impinging of calcified spicules on to the peritoneal covering. In the latter case, the white spikes of lime salts can sometimes be seen lying naked, pushing their points through a surrounding pink or red areola; the spikes are often 'flaked' with red shreds of organized lymph. Tenderness is *always* present, absolutely localized to the area of the pain.

Many of these cases are called 'neurotics'. They are a misery to themselves and to their doctors. Operations are performed with a diagnosis of appendicitis, and a tiny opening is made through which the tip of the cæcum is produced, the appendix is removed, and a beautiful scar is left. Any subsequent pain is said to be due to adhesions, and sometimes further operation is performed for the division of these. Massage is a favourite device; naturally it is a very painful business, but the patient (being neurotic!) is advised to persist with it. Relief can only come by a very prolonged process of natural cure or by the removal of the offending source of irritation by operation. Diagnosis can be made in nearly all these cases by X-ray examination.

It will be useful here to recapitulate what has been said by numerous authors on the symptoms and diagnosis of the condition, with special reference to its simulation of appendicitis.

Risley[1] says that the clinical picture is that of an acute right lower quadrant condition simulating appendicitis. There are two clinical types: (*a*) Distinctly palpable masses of glands with vague or no abdominal symptoms; and (*b*) Cases with alarming abdominal symptoms and signs developing suddenly when there are no palpable glands demonstrable. Diagnosis is often difficult or impossible.

Symptoms: (*a*) Trivial, fleeting, and not severe right-sided or general abdominal pain with or without disturbance of digestion and bowel regularity, the general health being good, or patient of a pale, rather sickly type. (*b*) More or less constant fairly localized abdominal pain, with a soreness referable to the cæcum. (*c*) Sudden (or gradual) onset of generalized, then localized, pain in the right lower quadrant. Temperature 99°, 100°, or 101°; nausea, vomiting, constipation or diarrhœa, with local tenderness, spasm, and resistance; white count 12,000 or 15,000; very sick appearance. There may be distention.

The process may be fulminating, and an immediate operation may save patient from general tuberculous peritonitis.

Carson[2] says the condition is probably much more common than is supposed, most cases being labelled 'indigestion'. He describes under-developed patients, not gaining weight and strength, never been strong; thin, listless, appetite poor, tongue furred; only a few cases of rise in temperature (99° to 100°); pulse only quick in pain. The main sign is pain—*absolutely typical* in character—"Sudden centralized abdominal pain severe enough to make the child cry, lasting fifteen minutes or less, relieved by pressure and hot applications, recurring perhaps two or three times a day, and stopping as suddenly as it began, so that in the intervals the patient is quite free". He thinks it is a true colic. He believes the gland stimulates the neighbouring vagus nerves proceeding to Auerbach's plexus, and causes a tonic contraction of the circular muscle-fibres of the small intestine, and

that the pain is due to this cause. In three cases he saw an intussusception. Can this cause the pain?

CARSON[2] also says it is rare to find any lump; there is no free fluid, no rigidity, no distention. In a few cases he has had an X-ray, but only once has he seen a vague shadow of a caseating mass.

CORNER[3] says that the children suffer from ill health, and abdominal pain referred to the umbilical region. These pains come on at night, and sometimes after food. There is loss of appetite, anorexia, often accompanied by some disturbance of the action of the bowels, more often inactivity and constipation than looseness of the bowels.

STRUTHERS[4] says:—

"Within recent years a number of cases have come under observation in which patients apparently suffering from appendicitis have turned out on operation to be suffering from lymphadenitis; that is to say, the appendix has been found perfectly healthy, and the only lesion detected has been enlargement of the lymph glands in the mesentery, with signs of peritoneal irritation over them. In most of the cases, but not in all, the enlargement has obviously been due mainly, if not altogether, to tuberculosis; but whether the acute symptoms simulating appendicitis have been due to the tuberculosis alone, to tuberculosis with super-added transient infection of another kind, or in some cases to non-tuberculous lymphadenitis alone, it is difficult and, in the meantime, almost impossible to say. . . . The attacks are relatively mild in character, with moderate fever and constitutional disturbance. The general appearance is not that of severe illness, and in particular the tongue is not heavily furred, nor has the breath the peculiar and almost characteristic odour associated with appendicitis. As a rule the acute symptoms tend to subside in a day or two if the case is watched. During the acute stage the glands are not often or readily felt, owing to the muscular resistance present. Under an anæsthetic, however, they may be more easily palpated, and when several are adherent to each other the resulting lump tends to be higher and nearer the middle line than the swelling caused by the inflamed appendix with omentum or intestine adherent to it. After an acute attack the persistence of a lump and its situation may clear up the diagnosis. It must be admitted, however, that a positive diagnosis as between appendicitis and mesenteric lymphadenitis often cannot be made. Appendicitis is a treacherous disease, and in doubtful cases a 'wait and see' policy is apt to be followed by disastrous results, especially in children. One thing may be asserted with confidence, namely, that opening the abdomen in cases of lymphadenitis does not appear to influence the glandular affection unfavourably. The patients do very well, and I have not yet seen a case in which apparent extension of the tuberculous disease has followed operation."

PARKER[5] says:—

"Unfortunately the diagnosis of this disease is very difficult, and in some cases even impossible, as it apparently follows no definite peculiar symptom-complex. A certain proportion of cases complain of vague abdominal pains pointing to no particular organ, in children possibly coming on at night or after the ingestion of food, accompanied by the loss of appetite and disturbance of bowel function, more often constipation than diarrhœa. . . . It is a peculiarity of this lymphatic gland disease that it occurs either with moderate pains pointing definitely to no abdominal organ, or appears, and with a certain preference, under the aspects of two of our worst and most frequent abdominal diseases. . . . Maylard says: 'When a palpable tumour is present, the diagnosis is easier than in adults, for where in the latter there are many conditions giving rise to tumours, simple and multiple, within the abdomen, in the former there are but few; indeed, it may be said that hard, movable lumps in the belly of a child, which are not fæcal and which remain constant under all conditions, are most certain to be tubercular mesenteric glands'. . . . So it may be truly said that because of the rarity of its appearance, the absolute lack of all indications of tuberculosis in other parts of the body, especially of the intestines, and its peculiar character even in the cases in which a mass is to be plainly felt, a diagnosis can be arrived at only in a conjectural way. In cases of the nature of

those reported, however, from the standpoint of treatment, the more or less accidental pathological diagnosis is not of decisive importance, as laparotomy is indicated anyway, and the condition found can be dealt with as indicated."

MAUCLAIRE[6] says of mesenteric tuberculosis of the lymphatic glands :—

"This form, which is so frequent with children, and constitutes tabes mesenterica, has given rise to some tentative operations. This lesion is mostly the result of a chronic evolution. Sometimes, however, it presents acute symptoms which simulate appendicitis, intussusception, etc. Sometimes these inflamed glands suppurate and discharge into the peritoneum or the intestine, which they infect secondarily. In an exceptional case their calcification led to a diagnosis of calculus in the common bile duct."

H. TYRRELL GRAY[7] says of the pain :—

"Inflammation of mesenteric glands, whether acute or chronic, may be associated with colic, which nearly always arises in the small intestine or the ileocolic angle. The primary focus in the intestine may itself be responsible for the pain, as already described ; but the glands themselves may also be responsible. For the inhibitory segment of the peristaltic wave normally exerts a physiological degree of tension on the mesentery during its passage, and, in the presence of inflamed glands, drags on these and causes pain."

SIR JOHN THOMSON-WALKER[8] refers to the frequency of the condition and the rarity of its diagnosis clinically. He states :—

"In 254 necropsies on tuberculous children at the Children's Hospital, Great Ormond Street, 151, or 59 per cent, had tuberculous mesenteric glands, whilst clinically only 46 cases were diagnosed in 6000 or 7000 patients at the Evelina Hospital.

"In a number of the 42 cases other pathological conditions were present in addition to the calcified abdominal glands. Thus in 7 cases there was stone in the kidney or ureter, in 2 there was pyelitis, in 3 urinary tuberculosis, and one patient was pregnant. These conditions all tended to mask the symptoms that might have been due to the glands and thus to confuse the diagnosis. But there were 28 cases in which no other disease beside the calcified abdominal glands could be detected, and the following notes in regard to symptoms are based upon these cases.

"Pain is the chief symptom in calcified abdominal glands, and in my cases it was the prominent feature in 25 of the 28 cases. It was a dull ache in 4, very acute pain amounting to abdominal colic in 14, and moderately acute in 7. In the majority of these pure cases the pain was a severe colic (14 in 25). The duration might vary from a few minutes to several hours. The pain commenced suddenly and usually ceased suddenly. In severity it was comparable to the two chief abdominal colics, namely renal and biliary colic, and was much more severe than that of appendicitis. In distribution it resembled that of moderate renal pain or of renal and ureteral colic in 17 cases, of appendicitis in 4, and the pain resembled biliary colic in one. In 5 the pain area was not defined, and in one case there was no pain. There were certain negative points that helped to distinguish the pain due to calcified glands from that of renal colic. Movement had practically no effect in initiating or in increasing the pain. Vomiting, so common in renal and also in biliary colic, was absent in these cases. There was no retraction of the testicle, and no pain referred to other parts of the body. There was very rarely any disturbance in the action of the bowels, such as might be expected in a case of appendicitis of long standing.

"A tender spot was present in the abdomen in four cases in which the calcified gland was the only disease present. The tender area lay directly over the calcified gland, and in all cases lay within the area of tenderness present in cases of appendicitis, so that this sign tended rather to confuse than to clear the diagnosis. In one case the calcified gland could be felt as a nodule, and rolled beneath the finger at a point in the line of the ureter above the brim of the pelvis.

"The proportion of cases in which the pain resembled that of renal pain, or colic, may have been unduly large from the fact that this class of case was the most likely to come under my care. The explanation of the pain is, I think, to be found in the proximity of the calcified glands to the ureter. The drag or presence of such

a calcareous mass would very easily cause ureteric spasm. The aching pain in the appendix region in 4 cases was due to the position of the calcified gland in this area.

"Blood was present in the urine in microscopic or in naked-eye amount in 6 cases in which no disease except the calcified gland could be detected.

"Any statement in regard to the relation of hæmaturia to calcified abdominal glands must be purely speculative. In the cases in which this symptom has been present, and no other cause has been ascertained, removal of the calcareous masses has been followed by disappearance of the hæmaturia.

"One may, therefore, I think, be justified in suggesting some relation of cause and effect between the glands and this symptom. The close anatomical relation of the calcareous glands to the ureter is undoubted. In several cases of calcareous glands I have found the passage of the catheter up the ureter has been arrested at the level of the glands, and although other more rigid or smaller catheters pass on, I have gained the impression that some pressure or drag existed at this part.

"Looking back on cases of obscure hæmaturia, I can recall, and I think most urologists must have seen, cases in which the only cause of the hæmaturia was some condition outside the ureter, such as an appendix abscess. One cannot avoid the conclusion, therefore, that calcareous glands may, by pressure or by dragging on the ureter, be the cause of hæmaturia."

DIAGNOSIS.

Risley[1] states that in 65 cases at the Massachusetts General Hospital, where tuberculous glands were found at operation and not diagnosed before a post-mortem, clinical records showed nothing whereby diagnosis could have been made. Many had obsolete or quiescent tuberculous glands without symptoms. Palpable masses of glands are the only diagnostic sign.

Carson[2] thinks few conditions could cause similar symptoms, i.e., in uncomplicated cases; he thinks a chronic appendicitis might do it, but the pain would be less frequent, last longer, and might be localized; also more tenderness in appendix. Rectal examination: may feel acute appendix, stone in ureter (frequent micturition, X-ray, hæmaturia). Digestive disorders, intestinal parasites, and tuberculosis.

Corner[3] says von Pirquet's test for bovine and human tubercle bacilli is usually negative. He thinks some infections due to bovine and some to human, some to both.

Caird[9] says:—

"A correct diagnosis was not always made. Some cases with a relatively short history were not discriminated from subacute appendicitis; in others, the mimicry of malignant disease was so perfect that the doubt was only solved on histological examination of the specimen after operation."

Hollenbach[10] considers diagnosis very difficult; the most varied pictures of organic disease may be simulated according to the position of the lymphomata.

Opie[11] relies upon X-ray diagnosis.

A. T. Jones[12] says:—

"The diagnosis of this condition is usually not made before operation, as there can be no distinctive symptoms due to the glandular enlargement *per se*."

RESEMBLANCE TO AND SIMULATION OF APPENDICITIS.

John Fraser[13], under the heading of 'Ileo-cæcal Lymphadenitis', says:—

"There is frequently a preceding attack of diarrhœa or intestinal disturbance, and the association of this is important, because the resulting alteration of the intestinal flora is possibly the exciting feature of the process. Severe abdominal pain appears—there may be vomiting, though such is by no means constant; there is considerable general disturbance and fever. The symptoms rarely last for more than twenty-four to forty-eight hours, when they abate with characteristic suddenness. How is one to distinguish such a case-history from that of appendicitis? I confess it is often exceedingly difficult—sometimes it is impossible. One or two

features may be suggestive. In lymphadenitis the pain is local from its commencement; it is never referred. This is a point, however, which in young children it may be impossible to verify. The tongue usually remains clean, the attack subsides with characteristic rapidity; fever introduces the illness, while in appendicitis temperature is comparatively late in its appearance. It may be possible to palpate the enlarged tender glands. But I fully realize that the points I have mentioned, while difficult to verify, are capable of considerable variation, and, if your experience tallies with mine, the diagnosis of the great majority of these cases will be made after they have been operated on for what was considered to be acute appendicitis. In view of the danger in children of the 'wait and see' policy where appendicitis is concerned, it is better that things should be so."

HOLLENBACH[10] states that sometimes acute and sometimes chronic appendicitis is simulated, both as regards history and condition. He says:—

"During the last few years we have observed six cases, of which four simulated acute and two chronic appendicitis. Without exception these were youthful patients. The chronic cases presented a true picture of a chronic appendicitis, both as regards history (as given by the patient: anamnesis) and condition. In acute cases the general health of the patient was proportionately little disturbed, with a temperature of 40°. There was great tenderness in the cæcal region on palpation, but no definite tension of the abdominal walls, and the pulse-rate was only slightly increased. It follows from this that the height of the temperature contrasts somewhat with the general and localized condition of the patient, and this is an observation which may be eventually taken into consideration when making an alternative diagnosis. Laparotomy revealed extensively swollen lymphatic glands in the ileocæcal mesentery, which in isolated glands attained the size of a walnut. Next to the calcified glands we found, in the case of acute cases, new swellings. The appendix had undergone no noteworthy changes—certainly not to the extent that it could be considered as the cause of the affection."

SCHMIEDEN[14] discusses the picture of tuberculous disease of the mesenteric glands in the ileocæcal angle, and describes it as typical, the knowledge of which is due to operative autopsies. In the large majority of cases it runs its course with symptoms which simulate chronic appendicitis. However, the alternative diagnosis of renal calculus, urinary calculus, gall-stones, or gynæcological trouble often has to be considered. Not infrequently, it causes severe attacks of an inflammatory nature accompanied by abdominal tension; at other times, again, symptoms appear which remind one of ileus. These misleading symptoms, accompanied by the periadenitis signs, are to be attributed to intestinal spasms.

ORTNER[15] mentions that in his book, *Clinical Symptomatology of Intestinal Diseases*, 1917, he has called attention to the fact (in several places) that tuberculosis of the mesenteric lymph glands may simulate acute as well as chronic appendicitis. He has shown the various points in the differential diagnoses, and has also discussed the fact that such a form of tuberculosis of the glands, just as in the cases of appendicitis, does not result in pain in the ileocæcal region but exclusively in the pit of the stomach, and therefore may simulate an appendicitis in this direction also. That such a form of tuberculosis in the ileocæcal angle may be recognized even before making an incision had once more been proved by a patient under his care, whom he handed over to a surgeon with the diagnosis of tuberculosis of the mesenteric lymph glands. The surgeon, when performing a laparotomy on this case immediately afterwards, found an acute tuberculous peritonitis, which had its origin in tuberculosis of mesenteric glands in the cæcal region.

ISELIN has reported eight cases of tuberculous mesenteric glands, six of which came to the operating table with a diagnosis of appendicitis.

KEPPLER and ERKES[16] say:—

"The symptoms picture of the cases diagnosed as appendicitis is as follows: The sudden commencement of vomiting and high temperature, severe pain with contraction or tension of muscles in the right side of the abdomen, as well as constipation. Thus there can be no doubt with regard to the diagnosis prior to operation. This accounts for the fact that in only one of our cases the use of the X rays was

resorted to. Tuberculous mesenteric glands may simulate the most varied abdominal affections. For instance, the caseous and calcified glands situated in the ileocæcal angle not infrequently simulate appendicitis ; those situated in the mesentery of the small intestine and in the transverse mesocolon occasionally simulate affections of the stomach or duodenum."

A. T. JONES[12] says :—

"Many of these cases have been operated upon with the diagnosis of appendicitis or some acute intestinal condition which presented a decidedly surgical appearance. Upon opening the abdomen, possibly a rather harmless appendix is found which is apparently not enough to account for symptoms. Further exploration in these cases reveals the mesentery studded with enlarged glands in various degrees of inflammation, and varying in size from that of a pea to that of a filbert. In some cases we may find large retroperitoneal masses composed of several glands, the whole mass being from the size of an egg to that of an orange or even larger."

BARRY and FENTON[17] say :—

"To surgeons who operate not infrequently for appendicitis, we feel sure in many cases it has happened they have cut down on appendices which they have been surprised to find to all appearance normal, or so little affected as to be obviously insufficient to give rise to the symptoms complained of. It is in such cases most searching investigation should be made for signs of tuberculosis in the cæcum, the small intestine, and their mesenteries ; should such conditions be found, an excision of the affected gut and its mesentery should be forthwith carried out. Even with an obviously diseased appendix, the surgeon should still prosecute a careful search for enlarged glands in the mesentery, and consider well the possible cause of the disease present, and how far simple appendectomy will really cure the patient ; for it is in tubercular affections that the one hope of a successful issue is to deal radically with the disease whilst in its earliest stages."

MACLAREN,[18] speaking of unsatisfactory appendicectomies, says :—

"Deaver, in his book on appendicitis, gives the impression that it is easy to make a differential diagnosis between chronic appendicitis and the neurasthenic, even when there has been no preceding acute attack. In Deaver's fourth edition, 1913, under the heading 'Recurrent Appendicitis', he says : 'The diagnosis of chronic appendicitis is usually clear. The plainer cases give a history of acute pain referred to the region of the appendix, and examination reveals definite soreness, and upon palpation over this region even in the absence of an acute attack a diagnosis may be made with almost invariable accuracy on this combination alone, i.e., pain and tenderness in the right inguinal region'." Maclaren thinks there is a real appendicitis and one he calls the neurasthenic variety. He says :—

"They, as a rule, are young women or girls from 12 to 24 years of age (occasionally in older women, rarely in men) who are not in good physical condition, have lost from ten to fifteen pounds in weight, are worrying over family or financial troubles, or over examinations at school ; they are of the flat-chested scaphoid abdominal type, and when it is necessary for them to stand a great deal they are flat-footed. They have pain in the head, back, pelvis, or abdomen, due to their general debility ; they complain of tenderness on pressure in various parts of the abdomen ; many of them complain of this tenderness mostly in the right inguinal region, and these are the ones who come to us for operation. But just as large a number come to us on account of abdominal discomforts after their appendices have been removed. These cases are frequently temporarily relieved, but in a few weeks their old symptoms recur. It has seemed to us that these neurasthenic cases are improved by our unintentional exercise upon them of the faith cure. For if we honestly believe that we have removed the cause of their distress, we do unconsciously exercise this power, whether we believe that we do or not. It has seemed to us that this pain and tenderness in the right inguinal region, in these cases, was usually due to the stretching of the duodenum from its fixed point under the liver to the ptosed pylorus in the pelvis. There is seldom any rise in temperature ; the pulse is normal as a rule ; occasionally in the younger patients it is rapid from excitement, probably due to the examination. There is no true abdominal rigidity,

and the pain in the abdomen in the chronic cases will be lessened or will disappear when the patient lies down. This last point has seemed to us to be perhaps the most important point in making a differential diagnosis between the true and the false appendicitis." He does not suggest that any are due to tuberculous glands!

DIFFERENTIAL DIAGNOSIS OF CALCIFIED MESENTERIC GLANDS BY RADIOLOGICAL EXAMINATION.

By L. A. Rowden, Leeds.

On radiological examination of the abdomen one frequently finds shadows in the lumbar or inguinal regions; these shadows are usually smaller than a threepenny piece, but occasionally they may be much larger. They are usually multiple, and seem to be somewhat more frequent on the right of the spine. Their outline is almost always irregular and indistinct, and their appearance mottled; by palpation they can be freely moved, and, when multiple, their relative positions can be altered. When viewed from behind they appear larger than when viewed from the front. On deep inspiration they move downwards for some distance and then take a marked inward direction towards the spine. During respiration their relative positions often change. Shadows conforming to the above characteristics are caused by calcified mesenteric glands. (*Fig.* 281.)

Renal or ureteral calculi may cause shadows in these regions, and the differential diagnosis may, in some cases, not be easy. The following points of difference should be noted: (1) Renal calculus is usually single, only about 10 per cent being multiple. (2) Their shadows are usually at a higher level. (3) They move downwards and slightly outwards on deep inspiration, and do not turn inwards towards the spine. (4) They cannot be freely moved by palpation. (5) They appear smaller when viewed from the back than from the front. (6) When multiple they keep their relative positions on respiration or palpation. (7) The shadows of urinary calculi have generally a sharply defined, regular outline, and are homogeneous.

Ureteric calculi are generally single, and lie within a line joining the tips of the transverse processes of the lower three lumbar vertebræ. They cannot be moved by palpation, and do not move on respiration unless high up.

Pyelography and the opaque ureteric catheter may be of considerable help in some of the more difficult cases. Phleboliths, and the teeth or bone of dermoid cysts, are usually well down in the pelvis, and need hardly be considered in the differential diagnosis.

Radiography alone is not sufficient in many cases for the correct interpretation of the shadows. Radioscopy is essential, and, if the foregoing points are carefully looked for, a correct diagnosis is easy in most cases.

MACROSCOPIC PATHOLOGY.

With regard to the macroscopic pathology of the condition, as seen by the surgeon, there are at least five varieties.

Stage 1: the stage of infection, analogous to the stage of simple adenitis, e.g., cervical, which subsequently becomes tuberculous, evidenced by an

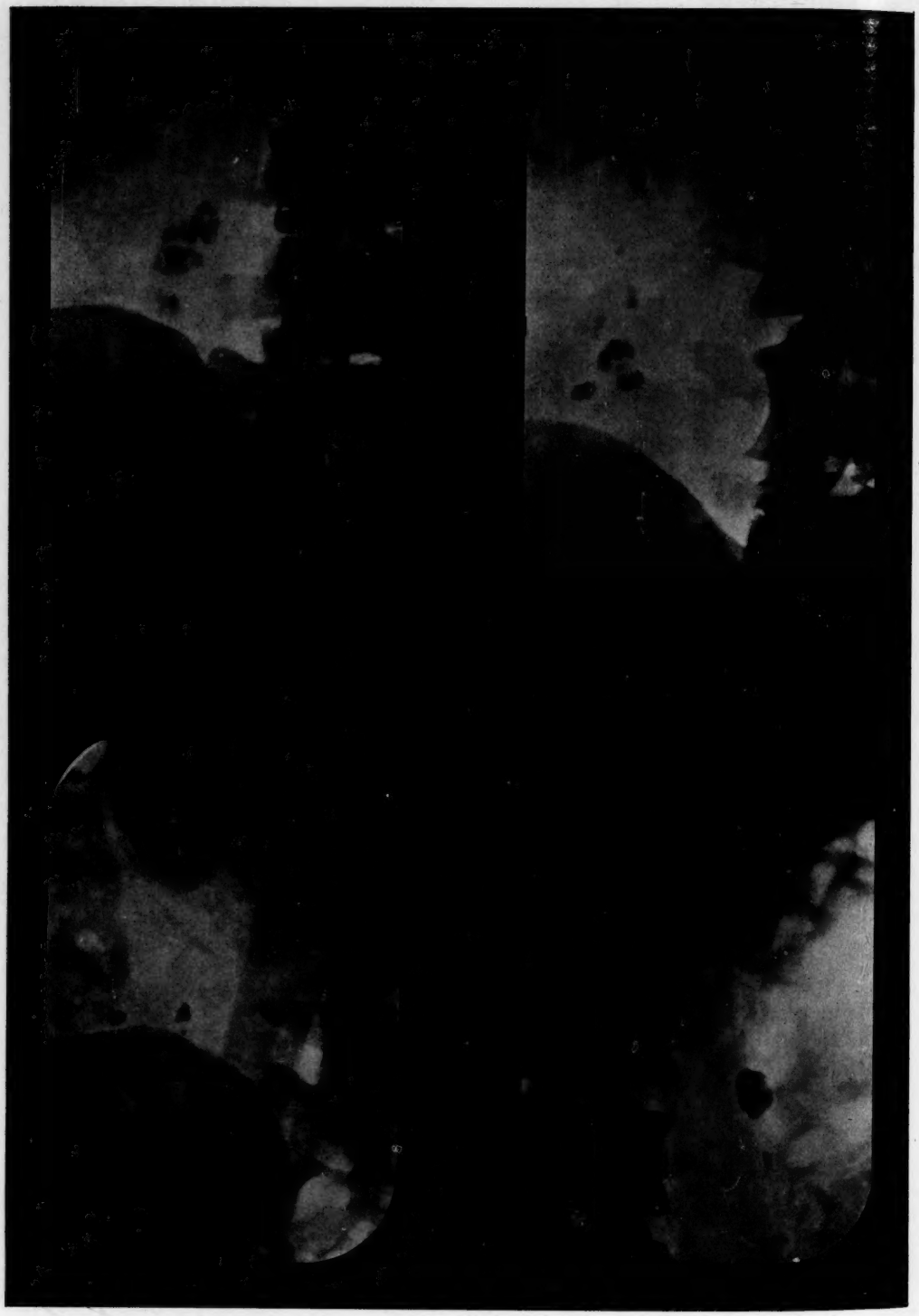

FIG. 281.—X-ray photographs by Dr. Leo Rowden.
The lower right-hand picture is transposed, and represents the right iliac fossa, not the left.

enlargement and a dull 'liver' redness of the gland or glands. This is the stage of simple adenitis, simple in that there is no naked-eye appearance of the tubercle.

Stage 2: the stage of 'spotted caseation', where tiny yellow spots, two, three, or four, appear in the reddened gland (*Fig.* 282). With this change there is frequently an extension of the inflammatory area from the gland to

FIG. 282.—*Stage* 2. Spotted caseation of glands.

the overlying peritoneum; assuming the disease is stayed, the yellow areas may become white (calcified) and give rise to

Stage 3: the stage of spotted calcification. In this stage there is more local irritation, due to actual irritation of the overlying peritoneum from spicules of calcified material, which may actually penetrate it. If this happens, each spicule becomes tipped by red flagellæ (organized lymph), and the base

of each spike where it penetrates the peritoneum has its own areola of redness. It is perfectly obvious how irritative a focus this must be, and how likely to cause localized pain. This stage is associated with exudation of fluid in small quantity into the peritoneal cavity.

Stage 4 may be termed the stage of massive caseation, where the whole gland becomes yellow. There is less liability to irritation of the overlying peritoneum, because the surface is smooth, with a string of vessels running in from borders. This type of gland may become as big as a Tangerine orange, and indeed in process of time tends to become pedunculated. It may

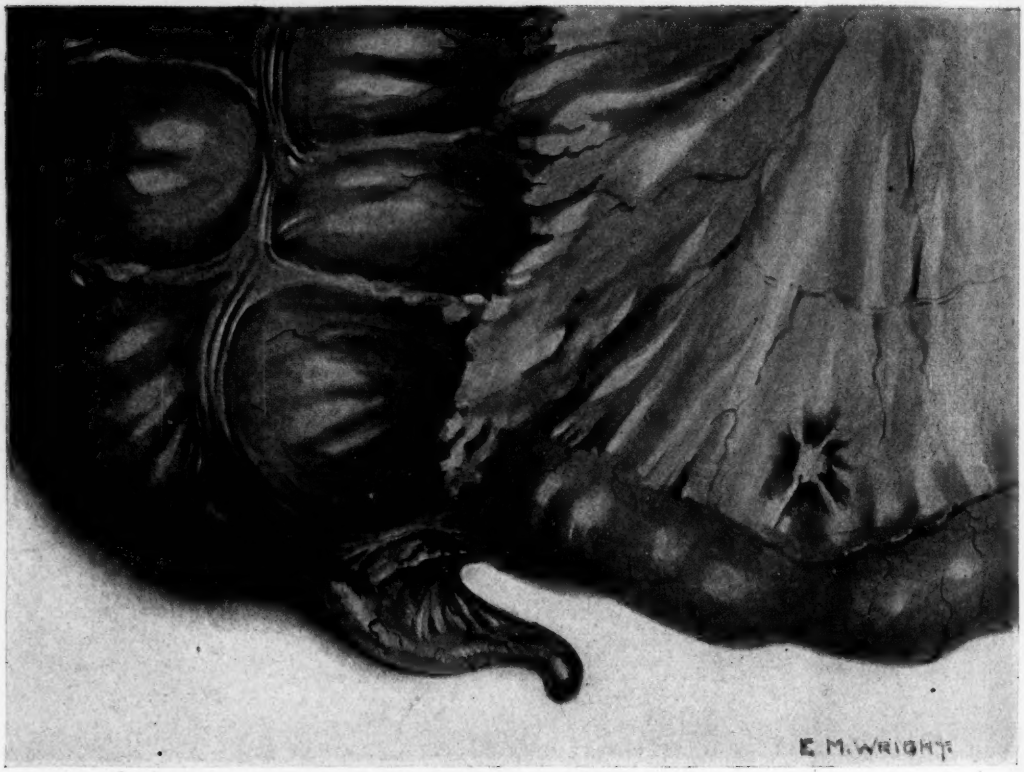

FIG. 283.—*Stage* 5. Shows a single cicatrized calcified gland, with surrounding red areola, and spasm of contiguous intestine.

even separate completely and be found lying loose in the abdominal cavity. As a rule a big caseous gland is solitary and gives rise to few symptoms. Whereas the contents of a calcified gland are sterile, an occasional tubercle bacillus is to be found in the caseous one.

By a process of further defensive change, massive calcification (*Stage* 5) may occur, but the resulting mass tends to cicatrize more than a caseous gland; it is therefore a small, irregular, intensely hard nodule with a much reddened areola flaked by red masses of lymph (*Fig.* 283). By reason of its physical characters it gives rise to a very serious degree of pain. Complicated

adhesions of omentum and intestine may give rise to large tumour formation and to serious intestinal obstruction.

TREATMENT.

As will be indicated, there are very varied opinions as to the proper means to be taken to achieve a cure.

Those cases in *young children* where an operation for acute appendicitis shows the source of the trouble to be an acute and possibly a non-tuberculous adenitis, and not an obvious appendicitis, are treated by appendicectomy alone. Where a *mass* of caseous or calcified glands is found, removal of the appendix and closure of the abdomen is indicated, with general treatment to follow. In young children where one or two glands only are seen to be the seat of caseation or calcification, one may consider removal of the diseased glands and of the appendix. One rule should be always borne in mind, i.e., never attempt removal of a diseased gland if it lies close to the mesenteric border of small or large intestine—a fæcal fistula may be the result. It would appear to be a dangerous thing to interfere in the way of either curettage of a gland or enucleation of it in the presence of an acute infection as indicated by a high temperature, though the presence of a localized peritonitis as indicated by a reddening of the overlying peritoneum should be no bar to its extirpation in the absence of severe general symptoms.

It should be borne in mind that there *is* a natural cure of the disease, and that such a cure will take place under favourable conditions in almost every case, though it may be a long time and it may be attended by attacks of pain. Appendicectomy should be done if the abdomen is opened at all, because, if it is not done, every attack of pain will give rise to alarming surmises that appendicitis is present.

If a diagnosis can be definitely made—usually by an X-ray examination showing calcified or caseous glands—operative interference may be inadvisable by reason of the large number of glands involved.

Where one or two calcified glands occur in adults and cause local symptoms, they should certainly be removed.

Removal of the affected gland or glands is easy so long as the gland is completely calcified or caseated, and so long as calcification has not produced rocky masses which, jutting out, entangle mesenteric blood-vessels. In the latter case enucleation may be very difficult. In a simple case, the left forefinger supporting the gland from behind, an incision is made at right angles to the line of the intestine through the peritoneum overlying the gland, by means of a dissector; the peritoneum is lifted from the front, and, in the case of the small intestine mesentery, from the back of the gland. After removal of the gland and careful ligation of every bleeding point, the peritoneum is closed by a row of Lembert sutures.

It is very often a difficult matter to remove a gland where the surface has merely become roughened by calcification—the overlying peritoneum cannot be stripped easily; in this case a tedious dissection may be necessary in order to avoid wounding vessels. It is often an easy matter to strip the upper surface, and, pushing the gland forwards with the left forefinger behind it so

as to make it pedunculated, place a pair of pressure forceps behind it in such a way as to catch the vessels running into it before cutting the gland away.

It sometimes happens that there is such a mass of inflammatory tissue in and around the capsule of the gland that excision cannot be done without a wide excision of mesentery, endangering the blood-supply of the bowel. In these cases one of two things may be done: (1) Opening and curettage of the gland, with careful hæmostasis and closure; (2) Sequestration of the gland by an omental flap or, better, an omental graft. It should always be borne in mind that the symptoms in the adult variety are due to a localized and probably only a traumatic peritonitis, and therefore a barrier of tissue between the mobile intestines and the irritating focus is all that is necessary.

As has been already said, active tubercle bacilli may be found even in completely caseous glands, whereas in completely calcified ones there are none. It would be obvious that, whatever local treatment be adopted, care should be taken to avoid infection as far as possible of neighbouring tissues, by packing and so forth. Injection of the glands with antiseptic fluid before opening them has been suggested by some authorities. 'Sterilization' of the gland cavities after curettage, by flavine and other fluids, has been largely done by the writer, and no case of exacerbation of symptoms has been encountered.

As in tuberculous peritonitis, opening of the abdomen alone seems to exert a beneficial effect, whether as a result of the congestion of the abdominal cavity and particularly of the portion handled, or as a result of the filling of the abdominal cavity with air, or, in acute cases, of the short temporary paralysis of the ileum and cæcum and the temporary fixation of the abdominal walls.

Wide excision of the cæcum with a portion of the small intestine has never been called for in the writer's experience, except where secondary involvement of bowel wall has occurred.

Whatever local treatment is adopted, it should be followed by *heliotherapy*, as advocated by Rollier at Leysin and by Sir Henry Gauvain in this country. By heliotherapy is understood treatment embracing not only exposure of the whole body to sunlight and clean air, but also the ingestion of plenty of wholesome food. It has recently been claimed that an 'artificial sunlight' produced by mercury vapour, tungsten arc, and other 'lights' which produce a special violet ray, is a very good substitute for the real thing. Rollier[19] gives an interesting historical account of treatment by heliotherapy.

Whatever form of tuberculosis be encountered, the question of *tuberculin* arises. In the cæcal glandular tuberculosis, tuberculin has not been shown to have any value. It is clear that in those cases where the symptoms are due to the presence of the residue of an old long-killed infection, tuberculin can have no value. But even when glands are only partly calcified or caseous, and where presumably there are living tubercle bacilli present, no effect has been noted as a special result of giving tuberculin.

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SOME CONSIDERATIONS OF THE OPERATION OF INTERNAL URETHROTOMY AND THE END-RESULTS THEREOF.

By G. P. B. HUDDY, London.

The 109 cases of internal urethrotomy performed at the London Hospital during the years 1921 to 1924 inclusive have been reviewed. These operations were performed on 107 patients, for in two cases the stricture recurred and a second operation was performed.

Introduction.—During the early years of the nineteenth century there were three methods in use for the treatment of impermeable strictures: the forcing of the stricture by a conical sound; repeated application of caustic; and perineal division of the stricture. It was during this period that a new principle of treatment was evolved. It was suggested by Howship, and later by McGhie,[1] that the stricture might be perforated by a sharp-cutting instrument passed through a cannula. The best known instrument of this type was that introduced in 1827 by R. A. Stafford,[2] surgeon to the Mary-le-Bone Infirmary, who used a lancetted stilette, contained within a silver catheter. His description of the operation runs: "The armed catheter is then passed down till the point rests against the stricture, and being held securely in such position, the spring is pressed by the thumb gently and gradually. As soon as any impression is made, the lancet should be allowed to retire into its sheath, and the blunt point of the catheter urged cautiously forward. If it do not pass on, the lancet may be again used as before." He was able to state in 1833 that he had performed this operation on forty to fifty occasions and there were no failures. Also there had been no instances of false passage, extravasation of urine, or hæmorrhage.

For the development of the modern operation of internal urethrotomy we are largely indebted to the French surgeons, who substituted a clean incision of the stricture for the brutal and non-surgical method of divulsion.

The early types of urethrotome incised the stricture from behind forwards, the most famous of these being the Civiale instrument. In order to use this latter it is necessary that the stricture should admit a F. 8 bougie. Civiale's urethrotome was strongly advocated by that great authority, Sir Henry Thompson, who pointed out that if preceded by preliminary continuous dilatation it could be used for even the smallest of strictures.

The year 1855 saw the introduction of the famous Maisonneuve urethrotome, which has since been modified by Teevan and Sir John Thomson-Walker. Since that date innumerable other types and modifications have been introduced both by surgeons and by instrument makers, but their popularity has been short-lived, and the modified Maisonneuve is the one most commonly in use to-day. This instrument was employed for the series of cases under consideration.

An important advance was made by Otis,[3] who, in 1874, devised an instrument for measuring the urethra—a urethrometer, by means of which he showed that the lumen of the urethra was much wider than had been supposed. Previously it was held that if a French 21 bougie could be passed the urethra could not be considered to be strictured; but he showed that the average urethra passed with ease a French 32.

Indications for and Contra-indications against Operation.—

Indications.—The indications for the use of internal urethrotomy depend upon the views of various surgeons. The main types of stricture which require urethrotomy are :—

1. The very narrow stricture which is only permeable with considerable difficulty and in spite of repeated efforts will not dilate readily.

2. The resilient stricture which dilates but as readily contracts.

3. The stricture in association with a perineal fistula, provided the fistula persists solely because of the presence of a stricture in front of it and not on account of a chronic infective condition.

Absolute Contra-indication.—Impermeability.

Contra-indications to Immediate Internal Urethrotomy (i.e., indications for the two-stage operation).—(1) Marked urinary infection; (2) Renal insufficiency; (3) Peri-urethral abscess; (4) Extravasation of urine.

The Two-Stage Operation.—The points just referred to as indications for the two-stage operation require further consideration.

1 *and* 2. *The Presence of Urinary Infection and Back-pressure Effects.*—These conditions usually go hand in hand. In 42 cases, none of which were fatal, albumin and pus were definitely noted as being present in the urine. Manifestly the presence of these two bodies in the urine is no absolute contra-indication to internal urethrotomy. Their presence, however, should act as a danger signal, and investigations be made as to the renal efficiency and possibly also cholesterol content of the blood. If these tests are unsatisfactory, then preliminary suprapubic cystotomy should precede internal urethrotomy, for free drainage is essential.

External urethrotomy combined with division of the stricture has been recommended by some authorities, but this is often a very tedious and time-consuming process, and so is obviously bad for such a patient. Moreover, perineal drainage results in increased fibrosis at this, the commonest, situation for a stricture.

3. *The Presence of a Peri-urethral Abscess.*—In such cases the abscess should first be drained, and when the wound is clean internal urethrotomy should be performed. This method was adopted in 5 of this series of cases, with very satisfactory results.

Peri-urethral suppuration is often held to be an indication for external division of the stricture. This would not appear to be the method of choice, because that mentioned above not only permits of free drainage, but also of a comparatively clean division of the stricture when all infection has subsided. If the abscess is opened and the stricture at the same time divided, infection is spread through the whole extent of the wound down to and including the urethral mucosa, with resulting additional fibrosis.

4. *Extravasation of Urine.*—This has been considered in a previous paper.[4]

The treatment recommended is primarily perineal drainage of the bladder combined with free incisions into the infected tissues, followed at a later date by internal urethrotomy.

The Operation and its Complications.—It is not proposed to give a full account of the operation in this paper. The method employed in every case was the ordinary Maisonneuve operation, immediately followed by dilatation of the urethra to its full calibre, and the tying in of a No. 12 catheter.

The complications met with in this series of cases are discussed below :—

1. *Urinary Fever.*—Rigors and pyrexia may occur at one or both of the following periods : (*a*) Immediately consequent upon the operation ; (*b*) Following upon removal of the catheter or dilatation.

a. Considering the first of these, rises of temperature are frequent, as will be seen from the following figures : No pyrexia, 49 ; Slight pyrexia (under 100°), 22 ; Pyrexia (over 100°), 28 ; Rigors, 10. The foregoing show that 38 of the 109 suffered from definite constitutional disturbance, while of the remaining 71, 49 showed no reaction whatever.

b. The second group consists of 29 cases, namely : Rigors, 12 ; Pyrexia, 17. Dilatation was a more frequent causative factor than the removal of the catheter.

R. Harrison[5] stated that rigors could be prevented in cases of internal urethrotomy by draining the bladder through a perineal incision. This immediately does away with some of the advantages of internal urethrotomy, for it prolongs convalescence and increases the difficulties of nursing, as urine drains through the perineal wound.

2. *Hæmorrhage.*—A certain degree of bleeding is an inevitable result of this operation, as direct means of hæmostasis cannot be applied. In no instance in this series was it of sufficient degree to cause alarm. In discussing this subject, in *Keen's Surgery*[6] it is stated : "The complications most often observed are extravasation . . . and, not rarely, fatal hæmorrhage from wounding of the dorsal vein, the plexus of Santorini, or the artery of the bulb." It would seem that the danger of hæmorrhage has been exaggerated, no doubt owing to the fact that on theoretical grounds it should be expected, because an incision is made into a highly vascular cavernous tissue. Sir Peter Freyer[7] reported several hundred cases of internal urethrotomy without a death, and very few attended by hæmorrhage of any importance. Sir John Thomson-Walker[8] also considered this danger exaggerated. Deaths, however, do occur from this cause. Hurry Fenwick[9] in 1888 recorded two fatal cases, not occurring in his own practice, and Myles,[10] ten years later, also recorded two fatal cases.

This harassing complication can in some measure be avoided—first, by holding the urethretome strictly in the mid-line so that the knife enters the interval between the two corpora cavernosa, and secondly by the tying in of a catheter. If the hæmorrhage is profuse, perineal pressure combined with the use of an in-dwelling catheter will usually suffice. The degree of distention of the bladder must be carefully watched, for as a result of the perineal pressure the blood, instead of appearing at the meatus, may pour into the bladder. If the bleeding still persists, drastic treatment must be adopted before the patient's condition is hopeless. External urethrotomy must be

performed at the site of the incised stricture, a petticoated catheter passed, and the wound packed firmly around it.

3. *Hemiplegia* (right).—This occurred in one case on the 4th day following the operation. A similar complication arising on the 6th day was recorded by the Marine Hospital Service, U.S.[11] following division of a stricture by the Otis urethrotome; the cause was found to be a hæmorrhage into the left corpus striatum.

4. *Atonic Bladder.*—One case occurred, which rapidly cleared up with regular catheterization. This, strictly speaking, is a complication of the urethral stricture rather than of the operation.

5. *Peri-urethral Abscess.*—This ensued in a case in which no in-dwelling catheter was used. The abscess was incised, and a second urethrotomy was required later.

6. *Epididymo-orchitis.*—Two cases occurred, in both of which a catheter had been tied in, for two days and four days respectively.

There was no case of urinary extravasation.

After-treatment.—Before discussing the results a brief indication as to after-treatment must be given, as the results are so entirely dependent upon this. As will be seen in a later paragraph, it is advisable to allow the catheter to remain in position at least four days. Four days after its removal the first dilatation is performed, up to at least E. 14/16, and in some cases even to E. 16/18. Provided no pyrexia follows, the patient may be allowed up, and discharged twenty-four hours later. Regular dilatation up to the sizes already mentioned must now be pursued.

The following table will serve as a guide to the regulation of the after-treatment :—

Intervals	No. of Attendances
1 week	4
2 weeks	3
3 „	3
4 „	3
6 „	2
8 „	1
10 „	1
12 „	1

Each interval is then increased by a month until the patient is seen twice yearly. In favourable cases the interval may later be increased to nine months or even a year, but the patient must always be kept under observation. The above is a guide only to be used for an average case, and must be modified as required for each individual.

Kollmann's dilator is a valuable instrument to use when the meatus is narrow or when over-dilatation of the stricture is desired.

For the early dilatations novocain should be used, for instrumentation is painful, and this is an important factor in influencing the regular attendance of the patient. Its use may be continued or not at the discretion of the surgeon.

The Post-operative Use of an In-dwelling Catheter.—This, which is an important feature of the after-treatment, has already been mentioned, and deserves further consideration.

The value of this procedure is denied by some surgeons (Coulson[12] and Sir Peter Freyer[7]). It has, however, the following advantages :—

1. By bringing a certain amount of pressure over the incised area it diminishes bleeding.

2. It conducts the urine away from the bladder, and as this urine is often infected, the risks of infection of the urethral wound and extravasation are thus considerably diminished.

3. If unsupported, the sides of the wound tend to fall together and the stricture tends to be re-formed (*Fig.* 284).

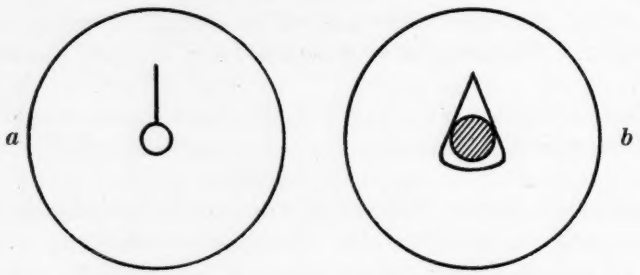

FIG. 284.—Showing incised stricture (*a*) without catheter, (*b*) with catheter inserted.

4. If no catheter is used, each dilatation must result in the tearing of young fibrous tissue binding the edges of the cleft, and so cause increased fibrosis.

In reviewing the 109 cases, there is no evidence of its having produced any ill effects.

Period during which Catheter should Remain in Position.—In comparing the post-operative pyrexia, etc., the following table was evolved, three groups of cases being chosen for the purpose.

PERIOD OF CATHETER USE	TOTAL CASES	NO PYREXIA	POST-OPERATIVE RIGORS	POST-OPERATIVE PYREXIA
No catheter	10	5	1	4
2 days	41	17	3	21
4 ,,	12	5	2	5
Totals	63	27	6	30

An attempt was made to estimate the end-result of these same cases, and the 4-day cases showed the best results ; but the figures obtained do not convey an accurate impression, because some patients attended for dilatation while others failed to do so. In considering whether the duration of the in-dwelling catheter may be of importance, the following points should be noted :—

1. One patient, mentioned below, in whom the catheter remained only two days, is attending the Stricture Department, and it is only with extreme difficulty that a 12/14 English bougie can be passed.

2. The process of healing commences early, as is shown by the frequency with which pyrexia and rigors follow the re-opening of the wound

by dilatation. A catheter, so long as it remains in position, prevents the cementing together of the raw edges by this rapid healing, and provided it causes no ill effects should be retained at least four days.

3. If the process of healing just mentioned occurs with unusual rapidity, it may result in rapid recurrence of the stricture. There was one such case in this series, in which a second urethrotomy was necessary a few weeks later. The catheter had remained in only two days. A somewhat similar case is that of the man, mentioned above, who developed a peri-urethral abscess and later required a second internal urethrotomy. It will be observed that an in-dwelling catheter had not been employed.

RESULTS OF OPERATION.

Mortality.—Two patients died following the operation. In the first of these, carcinoma of the bladder, vesical calculus, and purulent nephritis were associated with the urethral stricture. This case obviously can hardly be considered in estimating the immediate mortality. One case remains in which the death was consequent upon the operation :—

A male, age 52, was admitted with a long history of retention of urine with overflow. Internal urethrotomy was performed, and a catheter tied in for four days. Irregular pyrexia ensued, and one rigor followed the dilatation. Death occurred on the twelfth day. At necropsy the bladder was dilated, hypertrophied, and inflamed; the kidneys were hydronephrotic and showed a condition of pyelonephritis.

This case serves to emphasize the fact already mentioned, that where there is any doubt as to the renal efficiency this should be investigated and if necessary a two-stage operation be performed.

In a series of 1018 cases of internal urethrotomy at St. Peter's Hospital there were 8 deaths.[13] It will be observed that the mortality is roughly 1 per cent.

In all the cases reviewed in this paper the operation was a life-saving one, in so far as the patient was relieved of the prospect of the severe back-pressure effects of a very narrow stricture which resisted dilatation, and was not an 'opération de complaisance'. If the operation is performed on strictures of a wider calibre, the operative risk is obviously much less, for the likelihood of kidney failure or urinary infection is much diminished. This is shown in the teachings of Otis[3] in 1881, for he states regarding indications: "In my experience, the greatest number of strictures which call for operative measures are the so-called strictures of large calibre which produce and prolong urethral discharge". He then gives a total of 2163 operations by various surgeons without a death or permanent disability of any sort.

These results are so satisfactory, and especially so in the case of a wide-bore stricture, that the scope of internal urethrotomy should be enlarged, and it should be carried out in those cases which are only dilated with difficulty and by heroic persistence on the part of the patient and surgeon.

Late Results.—Subtracting the 2 deaths, 105 patients remain. These fall into the following classes: Died since discharge from hospital, 8; Traced, 78; Not traced, 19.

The 8 deaths all occurred in patients who failed to attend the Stricture

Department. *Cause of death*: Recurrence of stricture and kidney failure, 2; Cystitis and pyelonephritis, 1; Extraneous causes, 3; Cause not determined, 2.

The two cases of recurrence are very disappointing, for this is definitely preventable. It serves to emphasize the fact that internal urethrotomy of itself cannot be regarded as a curative operation. Scar tissue remaining must always be a potential cause of stricture, and the importance of regular post-operative dilatation cannot be too strongly impressed upon the patient and even the doctor. Following an internal urethrotomy, the urethra is usually enlarged even if after-treatment is not pursued, but in a few cases the additional fibrosis caused by division of the stricture results in further constriction.

Of the remaining 97 patients 78 have been traced, and the results are entirely satisfactory and the patients most grateful, many having expressed their thanks in eulogistic terms.

Of these 78, 44 are still under observation at the Stricture Section of the Genito-urinary Department, and have attended regularly. In this group the results can only be described as brilliant. All, with two exceptions, admit readily Thompson's sounds of the size English 14/16 or over; the intervals of dilatation have been prolonged up to many months, and in a few cases as much as one year. Of the two exceptions, the first is a patient who at operation was only dilated to E. 12/14 and now admits E. 13/15; the second is a man with a very dense tunnel stricture which can only be dilated to E. 12/14 with difficulty.

Information has been obtained from 34 who for different reasons are not attending the Department, some living at a considerable distance and attending their own doctors, a small number being afraid of dilatation. These patients, without exception, have expressed their complete satisfaction at the result. In 3 patients only has there been any recurrence of dysuria, but 9 others, although they state definitely that there is no difficulty of micturition, reply to another question that the stream is not quite as free as on discharge from hospital.

These results can be summarized thus:—

Attending the Stricture Clinic—results brilliant	44
Not attending the Stricture Clinic—quite well	22
" " " " " —some dysuria	3
" " " " " —no dysuria but flow not quite so free	9
	78

Of the patients in the last two groups, all but one have failed to pursue regular instrumentation. Once again, therefore, the great post-operative essential is regular dilatation.

CONCLUSIONS.

1. The operation should be preceded by suprapubic drainage of the bladder if there is evidence of renal insufficiency or marked urinary infection.

2. A peri-urethral abscess should first be drained, and internal urethrotomy performed when the infection has subsided.

3. Urinary extravasation is best treated by free incision into the infected parts combined with perineal drainage of the bladder, internal urethrotomy being performed at a subsequent operation.

4. The dangers of hæmorrhage have been exaggerated.

5. The results are extremely satisfactory, and the operation should be carried out in any case where dilatation is difficult or unsatisfactory.

6. An in-dwelling catheter has beneficial results, and should, unless any complication arises, remain in position four days.

7. Post-operative dilatation is essential.

It is with the greatest pleasure I record my thanks to Mr. Hugh Lett, Director of the Genito-urinary Department, and Mr. G. E. Neligan for permission to use their case records, and for their kindly and ever stimulating interest and advice in the work of the Stricture Section of the London Hospital Genito-urinary Department.

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THE APPLIED ANATOMY AND PHYSIOLOGY OF THE THYROID APPARATUS.*

By GEORGE SCOTT WILLIAMSON, London.

(Being the Arris and Gale Lecture delivered before the Royal College of Surgeons of England, on Feb. 18 and 20, 1925.)

INTRODUCTORY.

In presenting some new aspects of the anatomy and physiology of the thyroid apparatus, I wish to indicate how these new facts can assist the clinician in arriving at a more rational understanding of the relation, one to another, of the groups of diseases of the thyroid gland, in the hope that a more critical study of the symptoms presented by these diseases will ultimately lead to an understanding of the part played by the thyroid gland in the metabolic turnover. The subject, I think, is one that will repay close study by team workers. There is a tendency to discredit the anatomist and to exclude him from these teams. It is forgotten, perhaps, that the design and architecture of any engine is very completely controlled by the nature of the fuel and the manner in which that fuel is to be transformed into work. An engineer presented with a piston or a cylinder, or other part—even a damaged (pathological) member—of the engine, can very accurately deduce the nature of the fuel and the method of burning it. We are only beginning again to study anatomy from such a point of view, and already this is yielding results, as may be seen in the work of Aschoff and his pupils.

The germ of this vital attitude to anatomy is manifest everywhere in the works of John Hunter. It has awaited the quickening that lies in the new ground of modern physiology, as exemplified in the remarkable books of the late Sir William Bayliss.† It has but a remote relation to the study of the effect of death on cells and tissues, and none to the classificatory treatment of pathology, as Bland-Sutton has pointed out in his treatise on tumours.‡ I propose, therefore, to look at the anatomy of the thyroid apparatus from this 'vital' point of view.

The subject matter of this paper represents the joint work of Dr. Innes Pearse and the author.

ANATOMY.

The Microscopic Anatomy of the Thyroid Gland.—I will begin with a brief outline of the microscopic anatomy of the thyroid gland. The detailed studies of the minute anatomy were published by us in 1923.[1]

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* From the Laboratories of the Royal College of Surgeons, England, and from the Dunn Laboratory, St. Bartholomew's Hospital.

† *Principles of General Physiology ; Interfacial Forces and Phenomena in Physiology.*

‡ *Tumours, Innocent and Malignant.*

In the fœtus the epithelium of the thyroid gland is arranged in the form of long columns of tissue. They run a straight course, or are coiled and folded on themselves. Epithelial columns of this nature occur in all normal glands, in both sexes, and at all ages. In the adult gland they constitute the so-called 'interfollicular' tissue of the literature. Microscopic identification of these columns in the mass of interfollicular tissue depends upon the existence of a system of microcapillaries occupying a central position in each column.[2]

In certain conditions of marasmus in man, and in animals also during starvation, the whole adult thyroid epithelium may take this form. As a

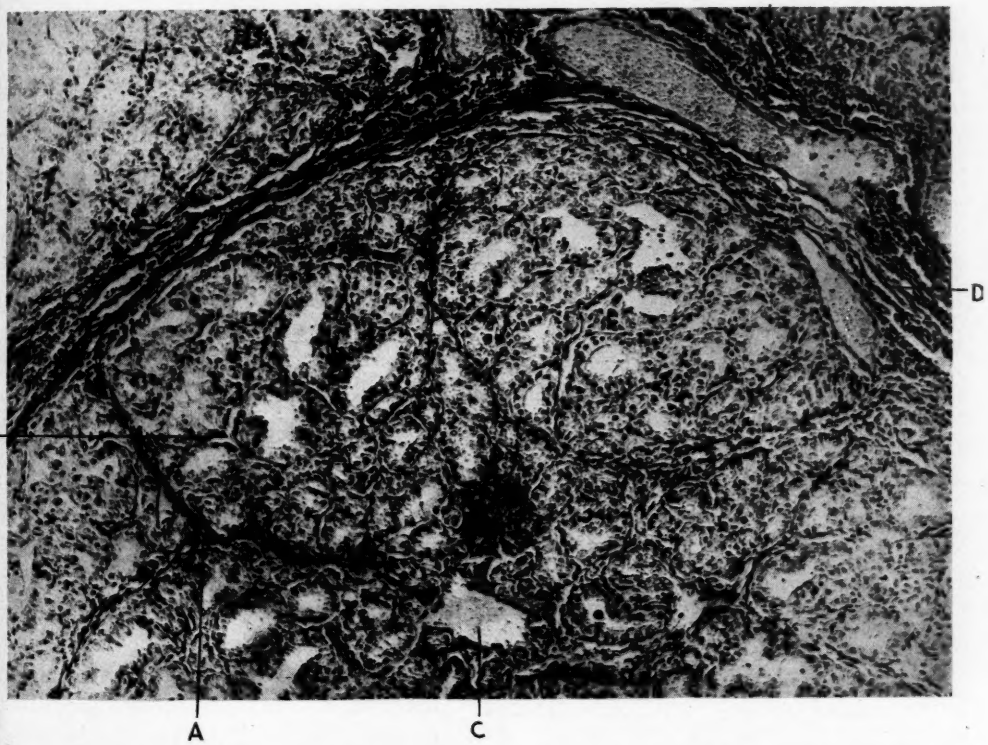

FIG. 285.—Microphotograph illustrating the gland-units within a lobule of the thyroid gland. This feature of the structure of the gland is only apparent when the tissue is engaged in producing secretion proper. The content of the follicles is not colloid. A, The thin fibro-elastic tissue defining the gland-units; B, The lymph sinusoid appearing as open inter-follicular spaces; C, The central intralobular lymph channel communicating with the lymph sinusoids; D, The interlobular fibrous-tissue stroma in which run the interlobular vessels.

rule, however, the solid core of the epithelium columns is replaced either by an accumulation of secretion on the one hand, or of colloid on the other hand. Although these events do not alter the essential structure of the columns, they do alter their appearance on section, and suggest a follicular nature for the tissue. This has led in the past to a description of the thyroid gland as being a collection of independent follicles, and it must be admitted that, were the phase of colloid storage alone studied, such a simple conception of the gland mechanism would appear to suffice. If, however, all phases of

function are to be reconciled, a larger area, and a design of greater intricacy than the simple follicle, must be ascribed to the unit of function in the thyroid gland.

The Gland-unit.—The simplest comprehensive grouping of tissues in the thyroid gland is the 'gland-unit'. A gland-unit consists of an endothelial-lined cavity enclosing coiled columns of epithelium accompanied by a basket-work system of capillaries. These capillaries enter the endothelial sac at its hilum, where they take origin from the intralobular capillaries (*Fig.* 285). The endothelial sac itself is an extension of the perivascular lymph channels which accompany the intralobular capillaries. The sac thus encloses a lymph space.

Such an association of structures recalls the liver. The gland-unit of the liver is an endothelial-lined sac, the portal blood sinusoid, in which lie columns of liver epithelium enmeshed in a plexus of Kupffer lymph capillaries.

In the thyroid gland, as in the liver, the endothelial sac is not always a mere potential space. Between the columns of epithelium, separating them one from another, there is often to be seen a thin, fluid non-colloid, lymph-like matter (*Fig.* 285). (*See also* Hürthle,[3] Matsunaga,[4] McCarrison,[5] and others.) Matsunaga had injected areas of this lymph sinusoid, which he calls a peri-epithelial space. Moreover, both in the human fœtus and the adult, this space is frequently occupied by lymphocytes. We have been able to trace the contents of the space to the perivascular lymph channels within the lobule, and beyond this to the lymph spaces of the interlobular interstitia, and thence to the hilum of the gland. Thus the sac in which the epithelial columns and their capillaries lie is an extension of the lymph system, and may be called a lymph sinusoid.

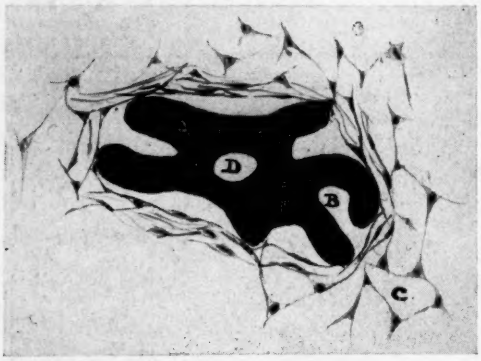

FIG. 286.—Drawing from human fœtal thyroid (*after Norris*), showing method of formation of peri-epithelial spaces into which grow the thyroid capillaries. A, Undifferentiated mass of multinucleated protoplasm—the thyroid *Anlage*; B, Peri-epithelial space; C, Interstitial cells giving origin to thyroid capillaries; D, A vacuole before rupture.

Embryology and Comparative Anatomy.—We may now turn to studies in the embryology and comparative anatomy of the thyroid for further evidence in support of such a construction in the gland.

The thyroid epithelium in the embryo appears first as an undifferentiated mass of multinucleated protoplasm ('*Anlage*') (*Fig.* 286). According to the latest work of Norris,[6] which we can corroborate, within this mass of protoplasm there appears a number of vacuoles; Norris, following the fate of these vacuoles, discovers that they rupture on the periphery of the *Anlage*. They thus form a series of peri-epithelial spaces between the thyroid *Anlage* and the surrounding connective tissue. Into the peri-epithelial spaces so formed the special thyroid blood-vessels grow and spread themselves over the

developing epithelium. Much later, in the column-like outgrowths of epithelium accumulations of secretion give a follicular appearance to the epithelial tissue. The peri-epithelial spaces of the embryo, therefore, would seem to be the forerunners of the lymph sinusoids of the fully developed thyroid gland.

Comparative anatomy affords similar evidence. We are deeply indebted to Mr. Burne, Physiological Curator, Royal College of Surgeons of England, for the favour of anticipating the publication by him of the following facts. By injecting and tracing the lymphatic system in adult angler fish (*Lophius piscatorius*), Mr. Burne has demonstrated the existence in the neck of a large ventral lymph sac* (*Fig.* 287). Our interest lies in the fact that in the wall of this lymph sac is set the thyroid gland. The thyroid follicles project into the lymph sac, or into bays formed by its reticulation (*Fig.* 288). The follicles are often stalked, floating at anchor in the fluid contents of the sac. Furthermore, the epithelium of the follicles is in direct contact with the contents of the lymph sac, except of course where the blood capillaries may intervene (*Fig.* 289)

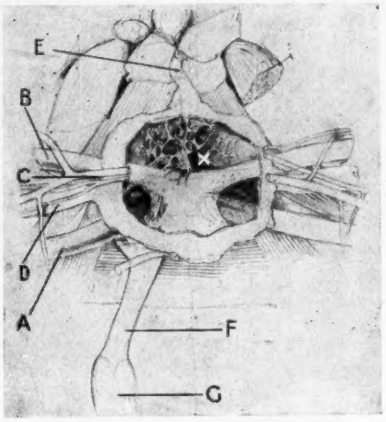

FIG. 287.—Photograph of drawing by Mr. Burne, R.C.S. Eng., of the thyroid body of adult angler fish, opened from the ventral surface, showing the central lymph sac, X, which has been opened to show the inner surface with its reticulations upon which lies the thyroid tissue. A, Hypobranchial artery; B, Afferent branchial artery II and III; C, Afferent branchial artery I; D, Lymph channel from ventral branchial sinus to the thyroid lymph sac; E, Lymph channel from ventral surface of intermandibular space; F, Lymph channel leading from thyroid sac to inferior jugular vein, G.

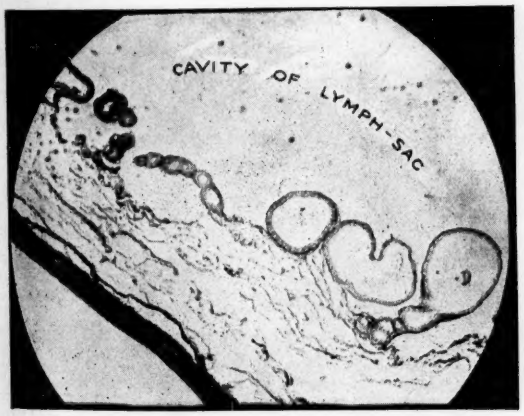

FIG. 288.—Wall of the ventral lymph sac of the adult angler fish, showing columns of thyroid tissue projecting into the lymph cavity and thus floating in the lymph itself. (× 40.)

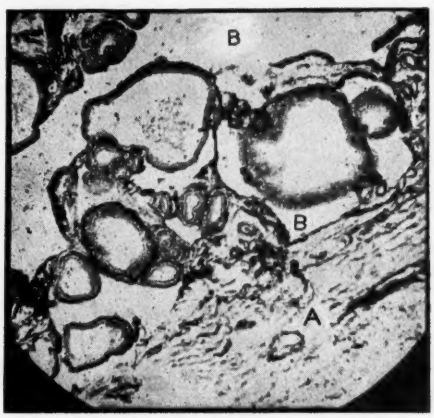

FIG. 289.—Wall of the lymph sac of adult angler fish, showing the naked thyroid epithelium lying in the lymph. A, Wall of sac; B, Lymph cavity. (× 65.)

* *See Specimen* No. A850A, Museum, Royal College of Surgeons, England.

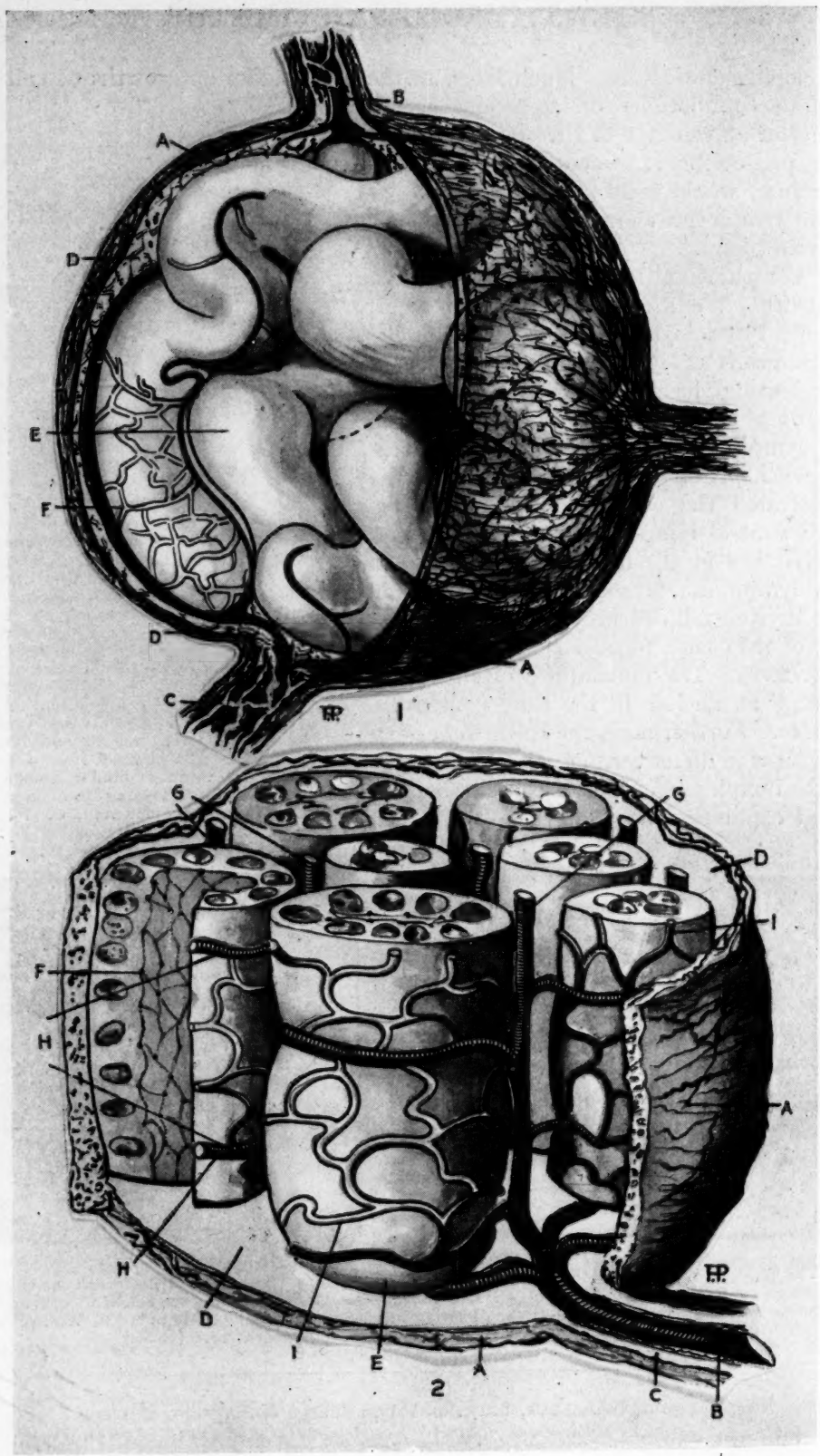

FIG. 290.

Thus embryologic and comparative morphology afford further substantiation of our concept of the thyroid gland-unit being fundamentally associated with the lymphatic system. Thus we conclude that *the morphological unit of the thyroid gland is a lymphatic sinusoid containing coiled columns of epithelium which are enmeshed in a basket-work of specialized blood capillaries* (*Figs.* 290, 291).

DESCRIPTION OF FIG. 290.

1. Diagrammatic representation of an isolated gland-unit of the thyroid organ. A, Fibro-elastic tissue capsule, partially removed to show the interior of the gland-unit; B, Intralobular blood-vessel, surrounded by C, the intralobular perivascular lymphatic plexus, which expands to become D, the serous cavity of the lymph sinusoid, in which lies E, the coiled and convoluted epithelium, enmeshed in F, the specific capillary plexus, which is an extension of the intralobular blood-vessels.

2. Schematic presentation of a section of a gland-unit, illustrating the blood-vascular arrangements. I, Anastomotic basket-work of capillaries stretched between H, the communicating capillaries which arise from G, concomitant capillaries which run in the length of E, the epithelial columns (seen in section) lying within D, the serous cavity of the lymphatic sinusoid. C, Intralobular lymphatics. B, Intralobular blood-vessels. A, Fibro-elastic tissue capsule of the gland-unit. (Diagram taken from the author's original paper in the *Journal of Pathology and Bacteriology.*)

Structure of the Lobule.—If the thyroid gland be constructed on the basis of the gland-unit, we find that a number of units are collected together by strands of interstitial connective tissue to form a lobule. The lobules are peculiar in that, although true blood-vessels (interlobular vessels) run in the interstitia surrounding them, all the intralobular vessels are simple endothelial vessels. They are provided with no support other than that of the fibro-elastic tissue capsule of the gland-units between which they lie. *It follows therefore that, within the thyroid lobule, artery and vein are indistinguishable.*

FIG. 291. — Microphotograph of the thyroid gland of a dog, taken from the work of Halsted, 1888. One gland-unit is seen surrounded by its fibro-elastic capsule. Three others partially occupy the field. All the gland-units are in the secreting stage of function. (×220.)

Furthermore, within the lobules there exists a central lymph channel common to the contained gland-units (*see Fig.* 285). This central lymph channel has much the same relation to the lymph sinusoid of the thyroid gland as the hepatic vein has to the portal blood sinusoid in the liver.

The lobules of the gland are bounded by an ample fibrous-tissue stroma in which arteries, veins, and lymphatics run to and from the lobules and their gland-units. This stroma knits lobule to lobule, thus forming the lobes of the gland.

The Hilum and Suspensory Ligament.—In each lateral lobe of the thyroid gland there is a clearly-defined hilum at which enter the arteries which supply the gland-units (*see Fig.* 310).

In most glands the hilum is a very clearly defined zone situated on the mesial or tracheal surface of each lateral lobe. It is set nearer the posterior margin of the tracheal surface of the lobe (*Fig.* 292). At the hilum a marked condensation of fascial tissue occurs, forming a firm band extending from the hilum to the thyroid and the cricoid cartilage. This ligament was described by Berry,[7] and shown by him to be of the nature of a suspensory ligament for the thyroid gland. Associated with this condensed tissue are the bundles of the recurrent laryngeal nerve running to their termination in the larynx. This ligament, together with some muscular slips on the anterior surface of the thyroid lobes, ensures the movement of the gland with the larynx, while at the same time it permits considerable movement of the gland on trachea and larynx. This latter movement is liable to early interference in malignant infiltrations, and in certain specific fibroses in pathological states of the thyroid gland.

FIG. 292.—Posterior view of right lobe of human thyroid gland *in situ* (half œsophagus removed), to show the structures in the hilum of the thyroid lobe. A, Parathyroid body; B, Thymic nodes; B2, Thymic node fibrosed; C, Laryngo-œsophageal branch of inferior thyroid artery; D, Anastomotic branch from superior thyroid artery; E, Parathyroid artery; F, Recurrent laryngeal nerve.

FIG. 293.—Drawing of an injection of the carotid artery in the human cadaver showing the free passage of the injection mass into the superficial thyroid venous plexus and thyroid veins. Note the insignificant branches of the superior thyroid artery passing to the fascial capsule, and the absence of any large branch of supply to the depths of the thyroid gland.

Thyroid Arterial Supply.—The functional arterial supply to the gland enters each lobe at the hilum (*see Figs.* 309, 310), from which it can be traced to the capillaries. This functional arterial supply is derived from the four or five terminal branches of the inferior thyroid artery. These are joined either by an inconstant anastomotic branch, or sometimes by a laryngeal branch, of the superior thyroid artery (*Figs.* 292, 310). As a rule the anastomotic branch is insignificant; in rare cases, it constitutes the largest source of arterial blood entering the hilum. *In any case, however, the blood supplying the functional gland-units enters the lobe at the hilum.*

What, then, is the purpose, for the thyroid gland, of the superior thyroid artery, apart from its insignificant anastomotic

supply to the inferior thyroid artery? Injection of this vessel indicates that it supplies twigs to the interstitial-tissue septa from the surface of the gland: these are largely distributed to the anterior and lateral surfaces of the gland (*Fig.* 293). The suggestion is that the superior thyroid artery has a certain nutrient function to the interstitial tissue, recalling the distribution of the hepatic artery in the liver.

There is to be noted one other peculiarity of the vascular supply. In nine cases out of ten, attempts to inject the superior thyroid vessels in the living animal as well as in the cadaver result in the injection mass readily passing to the larger surface veins (*Fig.* 293). It fails almost completely to pass through the intimate capillaries of the gland-unit. It would appear that there is a 'shunt circuit' in the interstitial tissue of the organ—a mechanism whereby physiological necessity determines the route traversed by the blood. It it as well to recall the weighty speculative conclusions that have been drawn from the measurement of the blood-volume issuing from or entering the thyroid arteries. Such measurements may refer to blood that has never been in contact with the thyroid parenchyma. This 'shunt circuit' may explain the comparative freedom of the gland from gross infection, and may account for the anomalous result of perfusion experiments.

As to the significance of this free arterio-venous anastomosis between the thyroid arteries and the voluminous thyroid venous plexus we venture no opinion. The old anatomists suggested a blood reservoir, and Barcroft's interesting work on the spleen,[8] and the recent observations on plethora (Trunecek[9]) may revive the speculation. There is still much to learn concerning the blood-vascular system of the thyroid apparatus.

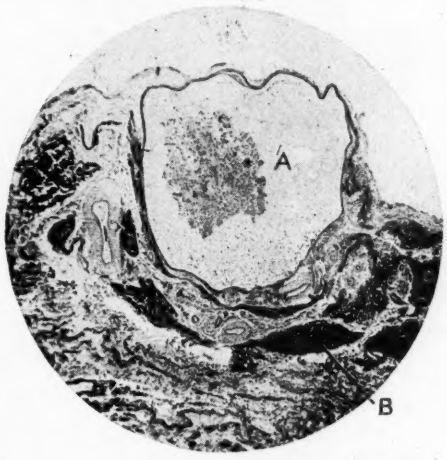

FIG. 294.—Cross-section of a dilated lymphatic channel (A) in the stalk of the adult human thymus. B, Thymic tissue. (× 35.)

The Thyro-thymic Lymph-vascular Connection.—Far more important is the lymph-vascular system of the thyroid apparatus. This deserves the closest study. It is a difficult subject, since complete injection of the lymphatics has not yet been achieved. Fortunately the continued activity of the gland itself provides us with a means, laborious in the extreme, of tracing the continuity, if not the course, of the lymphatic system. In a previous communication we pointed out the association of congestion of lymph in the thyroid sinusoids and lymph channels with similar accumulations in the thymic tissue and parathyroid bodies. This lymph has a tendency to accumulate in localized dilatations, or pseudo-cysts, occurring most frequently in the region of the hilum. Surgeons and embryologists are familiar with these endothelial-lined pseudo-cysts, which are so easily distinguished from colloid cysts by their steel-grey glistening endothelial membrane, and in

general by the fluidity of their contents (*Fig.* 294). Quite commonly, a series of cysts of this description can be followed almost in sequence from the hilum of the thyroid into the upper pole of the thymus along the so-called stalk, or ligament, of the thymic lobe (*Fig.* 295). Indeed, these varicosities often extend into the substance of the thymus in the mediastinum (*Fig.* 295). The region of distribution of the lymph-cysts coincides with the region of distribution of thymic tissue from the hilum downwards into the mediastinum. That isolated, as well as compact, masses of lymph tissue in this region are thymic tissue is demonstrated by the finding, even in the hilum of the thyroid lobe, of characteristic Hassall corpuscles.

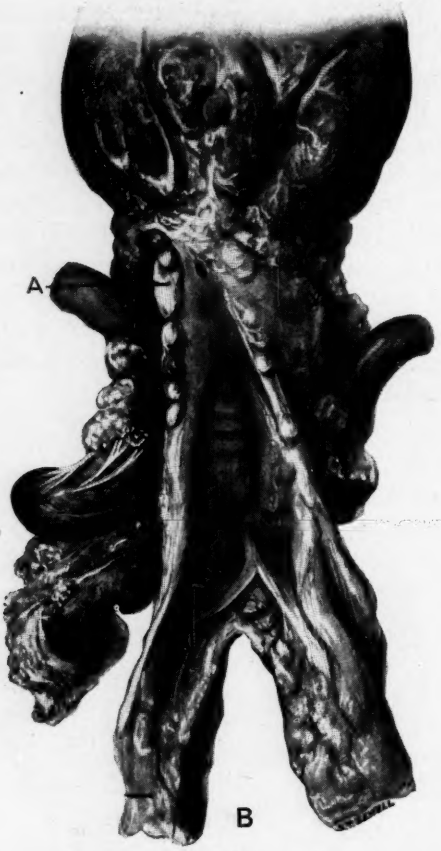

FIG. 295.—Drawing of thyroid and thymus showing the cord of lymphatics (Piersol's ligament) which extends between the two structures. Note the cystic distention on the course of the leash of lymphatics. Both leashes have undergone fibrosis, causing them to stand out as depicted here. A, Thyro-thymic ligament, showing a series of dilated lymph channels in its course; B, Lobes of thymus.

FIG. 296.—Drawing of dissection from fœtus, made by Sir Astley Cooper, showing the stalk of the thymus, which he demonstrated to be absorbent vessels connected with the thyroid lobes. (*Specimen* 3730 *D., R.C.S. Museum, London.*)

Careful dissection of the stalk of the thymus reveals the fact that it takes origin in the hilum of the thyroid lobe. This stalk, when it is well defined, is seen to be composed of vessels which have all the characteristics of lymphatic vessels (*Fig.* 296). Lymph is encountered in the channels. Frequently the component lymphatics, with care, can be dissected from the lateral surface of the trachea (*Figs.* 297, 298), upon which they lie, and traced to their origin in the hilum of the thyroid gland. The leash of lymph-vessels is always most prominent anteriorly, where it emerges from beneath the lower pole of the thyroid lobe (*Fig.* 297). On the other hand, these lymph-vessels may not be collected together into a cord or leash, but may spread

over the tracheal surface of the thyroid lobe and run down almost independently into the thymus. Thus it seems clear that there is a very definite lymph system which emerges at the hilum of the thyroid and passes directly into the lymph spaces of the thymic nodes. The sinusoidal lymph and migrating lymphocytes from the thyroid gland can at times be found in these lymph passages.

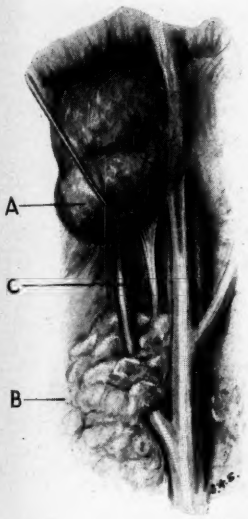

FIG. 297.—Drawing of leash of lymphatic vessels between the thyroid and thymus, dissected out to show its position on the anterolateral surface of the trachea, as it emerges from below the right thyroid lobe in its passage to the thymus. A, Right lobe of thyroid; B, Thymus; C, Leash of lymphatics.

FIG. 298.—Drawing of a dissection of the neck, showing thyroid and thymus *in situ*. The isthmus of the thyroid has been incised and the left lobe deflected from the trachea to expose the lymphatics passing from thyroid to thymus. A, Anterior leash of thyro-thymic lymph vessels; B, Upper pole of thymus; C, Body of thymus.

Confirmation of this special lymphatic system is obtained from 13 cases of cancer of the thyroid examined post mortem. In none of these cases were

the lymphatic glands of the neck, even those associated with the lower pole of the thyroid lobe, involved by the extension of the growth. In 9 of the 13 cases, however, a direct spread into the thymic tissue could be traced from the interstitial tissue lymphatics and from the invaded sinusoids (*Fig.* 299). This presence of a leash of lymphatics and thymic tissue in the fascia lying between the trachea and the thyroid has a most important bearing upon the direction of spread of cancer of the thyroid gland, and also upon certain other pathological conditions in which fibrosis may result in adhesions between the thyroid and the trachea upon which normally it moves freely.

It is generally accepted that, apart from these deep lymphatics, there are concomittant lymphatic vessels emerging with the veins on the surface of the gland. We have been unable to trace this system ourselves by the means at our disposal, but we do not doubt its existence.

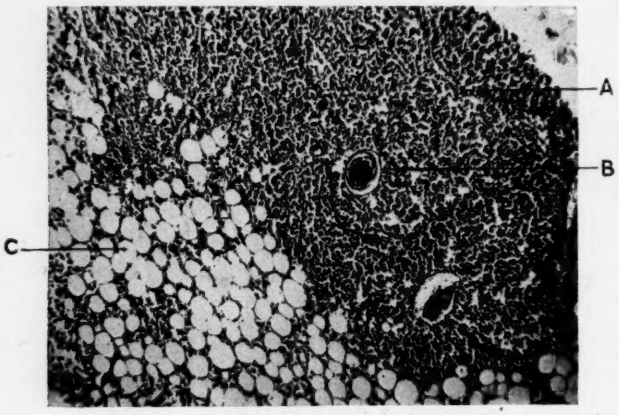

FIG. 299.—Showing the thymus invaded by cancer cells. From a case of carcinoma of the thyroid gland. A, Malignant cells; B, Hassall corpuscle; C, Fat tissue. (× 100.)

We are concerned only to point out that anatomical, physiological, and pathological evidence is available to show that *the thyroid and the thymus are directly and intimately connected through the medium of a system of lymph-vessels, which emerge from the region of the hilum of the thyroid and enter the thymus at its upper pole.*

Sir Astley Cooper,[10] speaking of the cervical pole of the thymus, says, "I have generally seen it joined by vessels to the thyroid gland". Elsewhere he defines the arterio-venous connection, and makes it clear that these uniting vessels are 'absorbent' or lymph vessels continuous with the central reservoirs or ducts of the thymus, and contain 'chyle'. In the calf he succeeded in some measure in injecting the 'absorbent' vessels, and to-day some of his specimens are to be seen in the Museum of the Royal College of Surgeons.* We are thus presenting a confirmation of work published as long ago as 1832 by a distinguished Fellow of this College.

THE THYMUS.

Microscopic Anatomy.—The thymus is a gland composed of endothelium. The endothelium is arranged in lobes or nodules, the individual cells of which also form the walls of a complex aggregate of venous and lymphatic capillaries. These lobules are held together extremely loosely in a definite capsule, very few strands of which penetrate into the substance of the

* Nos. 3730x, 3730xA, Museum, Royal College of Surgeons, England.

gland. In man, in these strands of interstitial tissue are to be found the typical Hassall corpuscles. The thymic gland as a whole has a very poor arterial blood-supply. Such is the basic structure of the thymic tissue.

FIG. 300.—Section through the body of the thymus. The mixture of vesiculated fat tissue and lymphocytes represents two phases in the physiology of the gland. (× 80.)

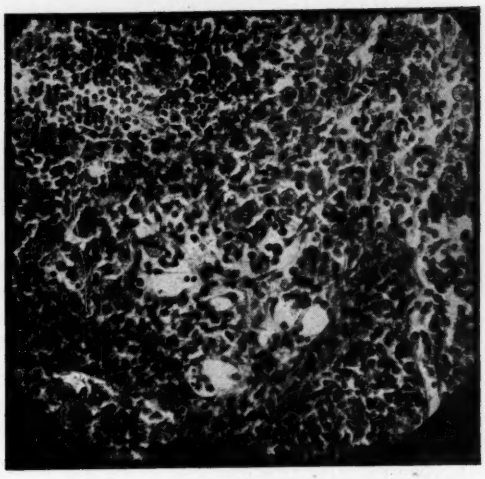

FIG. 301.—Showing the endothelioid or granular fat-phase in the human thymus gland. Note the large granular endothelioid cells and the relative diminution in lymphocytes in the surrounding tissue. (× 170.)

The thymus is modified as regards its appearance by the physiological demands made upon it. This liability to variation, or metatrophy, is characteristic of the thymus and certain other endothelial glands. The morphological picture of the metatrophy of the thymic gland exhibits three varieties: (1) A lymphocytic variation (*Fig.* 300); (2) A granular or lipoidal fat variation (*Fig.* 301); (3) A vesiculated fat variation (*Fig.* 302). These pictures of the physiological changes are similar to those described by Gulland[11] and later by Job[12] in the so-called secondary lymphatic glands. They describe the conversion of vesiculated fat tissue into tissue bearing lymphocytes, and even going on to form the endothelial germ centres under the influence of activity in the adjacent (preformed) lymph glands. It will be seen therefore that the thymic gland is not exceptional in manifesting great differences in appearance in the course of its activity.

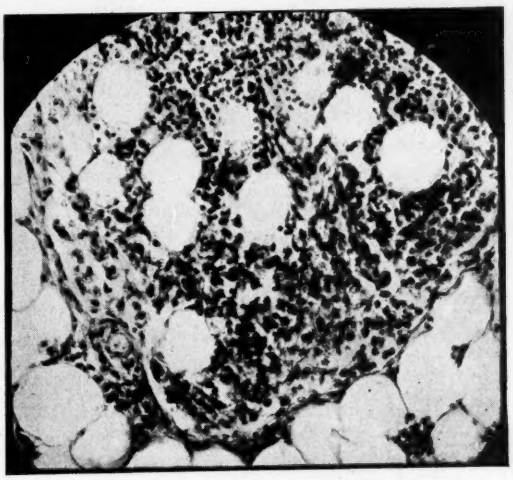

FIG. 302.—Sections through another part of the thymus showing the fat phase. Endothelioid cells and lymphocytes are relatively few in number, their place being taken by vesiculated fat. (× 185.)

The vesiculated fat phase is the one commonly found in the adult. This fact explains the common view that the thymus suffers atrophy in early life. Apart from this, however, Hammar[13] has amply demonstrated that the typical lymphoid phase can be found at all ages in the human thymus.

The thymus may thus be described as an endothelial gland peculiarly associated with the venous and the lymph-vascular system. From its dynamic features it is definable as a lymph fat gland.* We thus present a confirmation of the work of Kolliker, Toldts, Pende, and A. Kohn.[14] We have shown elsewhere that the lymphocytic activity occurs when the thyroid gland is most active,[2] and we look upon the thymus as a tissue which modifies the secretion, and stores any excess of secretion, of the thyroid gland.†

PARATHYROID BODIES.

The identification of parathyroid bodies is always a matter of difficulty. They are most frequently confused with nodes of thymic tissue (*see Figs.* 309, 310). The Hassall corpuscles, when present, serve to identify thymic tissue. We have found them present in nodes of tissue selected as representative parathyroid bodies. Except for the presence of the Hassall corpuscles, it is often difficult to distinguish between the endothelioid phase of thymic tissues and parathyroid tissue.

FIG. 303.—Drawing of a section of human parathyroid fixed and stained by the author's technique. The parathyroid body is shown together with the lymph tissue which by the presence of Hassall's corpuscles (A) can be recognized as thymic tissue. This was taken from the region of the hilum of the thyroid gland.

Parathyroid tissue is, in our experience, only typical when its tubular structure is apparent, when the tissue is compact, and has a definite arterial blood-supply. Apparently the parathyroid tissue can be scattered widely as microscopic fragments in the hilum of each thyroid lobe. When a parathyroid body is compact, the best guide to it is the parathyroid artery, which is a considerable vessel (Halsted[16]). Thymic nodes, on the other hand, have a trivial arterial supply. It is also to be remembered that the parathyroid tissue is often embedded in masses of thymic tissue, so that almost any well-formed nodule in the hilum of the thyroid lobe may prove to be parathyroid (*Fig.* 303).

As a compact body the parathyroid seems to be the site of termination of a direct branch of the sympathetic nervous system (*Fig.* 304). Usually

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* It is perhaps appropriate to refer here to the recent work of Cramer[15] and others on certain 'lipoidal fat glands', in that they present metatrophic features comparable with those found in the thymus.

† An alternative to this is that the thymus is the storehouse of 'raw material' for thyroid activity.

the branch is derived directly from the superior or middle ganglion, although it may reach the parathyroid body through the medium of the very complicated plexus situated in the fascia on the posterior surface of the thyroid lobe (*Figs.* 309, 310). However the branch arises, it is frequently ganglionated. The ganglion may be on the nerve as it enters the parathyroid body with the artery (*Fig.* 305), or it may be embedded in the capsule of the parathyroid body. The parathyroid body, therefore, like the suprarenal and pituitary bodies, seems to be closely associated with the sympathetic nervous system, and deserves special investigation from this point of view.

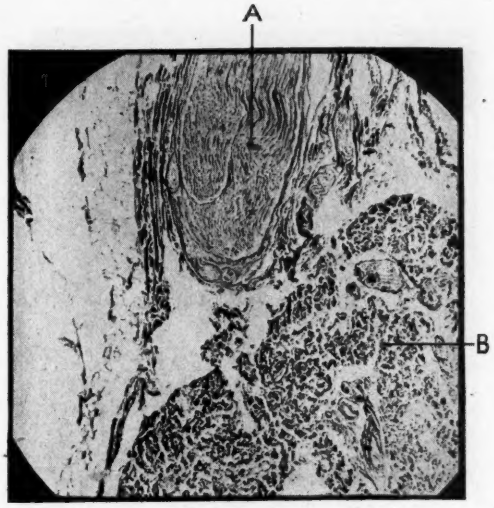

FIG. 304.—Showing a branch of the cervical sympathetic lying alongside the parathyroid body prior to the distribution of its filaments in the substance of that organ. A, Sympathetic; B, Parathyroid. (× 75.)

The parathyroid body has a second feature worthy of note. This is the essential tubular arrangement of the parenchyma. Under normal conditions evidence of this is found in an occasional tube containing the fluid, lymph-like material (*Figs.* 306, 307, 308). When this appearance is prevalent in the parathyroid tissue, it is definitely associated with secretory activity in the thyroid tissue, when the tissue is stained by the author's technique.[2] The same type of fluid, and occasionally lymphocytes, occupy both the lymph spaces of the thyroid gland and the tubes of the parathyroid body. In extreme degrees of parathyroid activity it would seem possible to trace the continuity of this fluid substance from the thyroid to the parathyroid. This fluid in the parathyroid body has in the past been taken to imply the identity of the thyroid and parathyroid epithelium. This we have already demonstrated not to be the case.[1] Further, in the literature it is generally conceded that the content of the parathyroid tube is not true colloid, but a thin granular fluid lymph. This we identify with *secretion* in the lymph spaces of the thyroid. Moreover, the parathyroid in man seems to be constantly associated with one or more thymic nodes which participate in changes occurring in

FIG. 305.—Human parathyroid artery (B), alongside which is the ganglionated branch of the sympathetic (A) going to the parathyroid body. (×50.)

the parathyroid. Extreme degrees of hypertrophic activity of the thyroid tissue (so-called hyperplasia) may coincide with the fairly complete conver-

FIG. 306.—Illustrating the general structure of the human parathyroid, showing the endothelial stroma enclosing the tubulate lobules of the parenchyma. (× 150.)

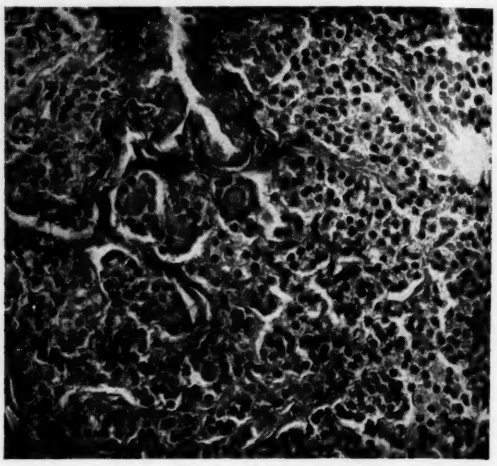

FIG. 307.—Human parathyroid gland, showing tubular nature of its structure. Two tubes are shown containing material similar to the secretion produced in the thyroid during secretory activity. (× 180.)

sion of the parathyroid body into a mass of follicular tissue (*Fig.* 308). This occurs in the embryo with great frequency, and in the adult occasionally, and it is a mistake to look upon these conditions as pathological.

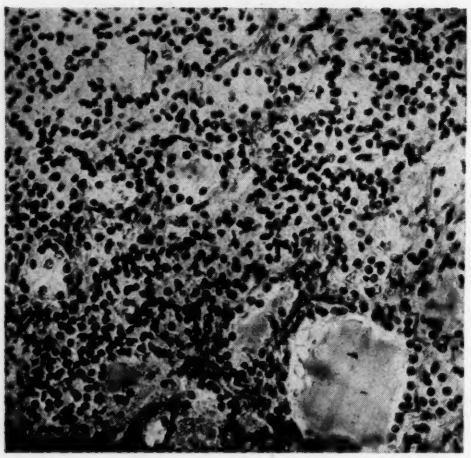

FIG. 308.—The same parathyroid body as in *Fig.* 307, stained by author's technique. The appearance of follicles and the refractility of their contained material suggest thyroid tissue, but the epithelial columns contain no central microcapillary system shown in all thyroid tissue stained with this technique. (× 185.)

It will be seen therefore that either the parathyroid tissue concurrently with the thyroid tissue accumulates secretion, or that the parathyroid body receives from the thyroid gland some of its secretion en route to the special thymic nodes to which the parathyroid is attached. We adopt the latter point of view, because *we have not been able to find fluid accumulations in the parathyroid in the absence of secretory activity in the thyroid gland.*

Further, lymphocytes are produced in the thyroid gland during secretory activity, and can be traced, via the perivascular lymphatics of the gland, into the tubular structures of the parathyroid body.

The conclusion we reach is that the parathyroid and thyroid glands work hand-in-glove in the execution of some common purpose.

SUMMARY OF ANATOMICAL CONSIDERATIONS (*Figs.* 309, 310).

1. A lymph sinusoid is described as the basic architectural feature of the thyroid gland. Its physiological morphology is explained.

2. The conception receives support from studies in embryology and comparative anatomy.

3. The functional arterial supply of the thyroid enters the gland at the hilum: it is derived from the inferior thyroid artery.

4. A subsidiary arterial supply (possibly nutrient) is derived from the superior thyroid artery, which usually contributes but little blood to the functional gland-units.

5. A 'shunt circuit' vascular mechanism is shown to exist in the thyroid gland.

6. A thyro-thymic leash of lymphatic vessels is demonstrated. This is shown to arise in the hilum of the thyroid and to pass down, on the anterolateral surface of the trachea, into the thymic lobes.

7. Lymph drainage of the thyroid is discussed, and it is suggested that the thymus gland is a reservoir for thyroid products.

8. The thymus is shown not to undergo atrophy in youth, but to persist and manifest a metamorphism co-incident with the nature of the demands made upon its function. It is analogous to other lymph fat-glands.

9. The parathyroid body is associated with a ganglionated branch of the cervical sympathetic nerve.

10. The parathyroid body is tubular in arrangement; at times the lumen contains material similar to the lymphlike secretion of the thyroid.

11. The probability is discussed of this material having been passed on to the parathyroid body from an actively secreting thyroid gland.

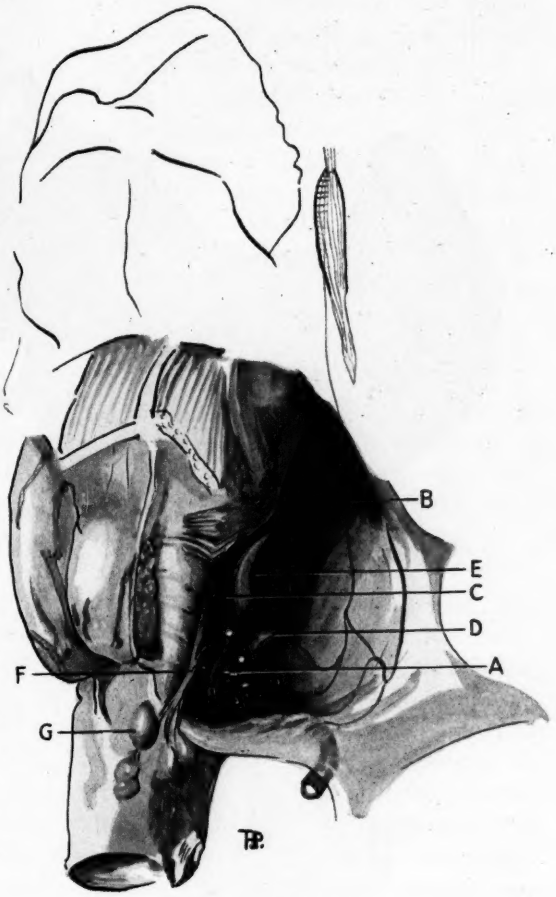

FIG. 309.—Drawing of the fascial bed of the left lobe of the thyroid gland to show the relative positions of the structures occupying the hilum of the thyroid lobe. A, Inferior thyroid artery; B, Branch of the sympathetic nerve; C, Recurrent laryngeal nerve; D, Parathyroid body; E, Lymph-fat gland body (thymic tissue); F, Thyro-thymic leash of lymphatic vessels; G, Thymic tissue.

PHYSIOLOGY.

In our earlier studies of the thyroid apparatus[1] we have shown that there are two processes that occur in the normal thyroid gland. One is the passive accumulation of colloid, or colloid storage, the other is the active process

of secretion. These are distinct functions, and colloid is a substance different from secretion.

Hürthle,[3] writing in 1894, recognized that there were two different processes in the epithelium, each one leading to the accumulation of material within the follicle.* Hitherto his original suggestion that these appearances

FIG. 310.—Posterior view of the thyroid apparatus, showing the relations and connections of thyroid, parathyroid, and thymus glands. Drawn from a dissection. A, The emerging plexus of lymphatics uniting thyroid and thymus; B, The inferior thyroid artery breaking up into its terminal branches in the hilum of the gland: note the cut œsophageal branch and the trivial anastomotic contribution from the superior thyroid artery: C. The inferior laryngeal nerve, showing its relation to the posterior border of the hilum of the lobe, the inferior artery and its branches, and the parathyroid body; D, Parathyroid body; E, Thymic nodes; F, Sympathetic nerves forming a plexus in the fascia over the posterior surface of the lobe, contributing twigs to the gland via the inferior artery, and a ganglionated branch to the parathyroid body.

could not be reconciled as the expression of one function has perforce been neglected, and the production of colloid has always been regarded as the end-result of all activity. As long as the simple follicle was accepted as the

* "Bei der Secretbildung sind nach meinen Beobachtungen zwei verschiedene Formen der Secretion zu unterscheiden, nämlich (1) Secretbildung des Follikelepithels mit Erhaltung der Zellen und (2) Secretbildung durch Untergang von Zellen".

unit of function, this was inevitable. When, however, the larger area of the gland-unit was recognized as the unit of function, it at once became obvious that colloid and secretion were different substances, and that colloid was not the result of activity in the tissue in their so-called 'hypertrophic' state.

The Process of Colloid Storage.—We have shown that the accumulation of colloid within the epithelial column can be followed from the first microscopic droplet to the distended vesicle of the normal gland in the colloid-storage phase of its activity. In every stage of colloid formation examined histologically, the epithelium, the sinusoid, and the endothelium of the gland-unit all maintained a perfectly uniform and constant appearance of quiescence. Furthermore, in a gland-unit in which colloid storage is in process there is no evidence of production of secretion. It would seem necessary for the epithelium to evacuate the stored colloid and return to the solid form prior to the production of secretion in that same gland-unit. This sequence of events was first observed in the experimental researches of McCarrison before being put forward by us.

The Process of Secretion.—In this process every element of the gland-unit takes active part. The epithelial nuclei, the cytoplasm, and the micro-capillary system all become laden with granules. This granular material ultimately liquefies, and occupies the centre of the epithelium, thus forming an accumulation or 'lake' of secretion in the column. At the same time the endothelial cells of the sinusoidal capillaries become laden with granules, and a thin fluid and lymphocytes appear in the sinusoid itself and in the perivascular lymphatics surrounding it.

The picture of a gland-unit in secretion is thus a totally different one from that of a gland-unit storing colloid. Next to a gland-unit which is in process of secretion there may be other gland-units in the process of storing colloid.[17] One gland-unit, however, is only occupied in carrying out one function at once. It may be argued that, although the two processes do not occur together in the same gland-unit, nevertheless the secretion formed in one may pass on to an adjacent gland-unit for storage in an altered form as colloid. We have not discovered in the gland any anatomical channels whereby such transference could be affected. It certainly does not occur through the medium of the lymphatic channels. The lymph channels and sinusoids never contain colloid during the process of colloid storage, while the fluid within the sinusoidal spaces during secretion has a similarity to secretion itself.

Significance of the Two Functional Cycles.—Colloid is not secretion. We have already set forth elsewhere[18] reasons for the probability of colloid being of the nature of a carrier or vehicle which, like the taurocholates and glycocholates of the bile, is continually in circulation in the body. Only the excess or reserve of bile is stored in the gall-bladder. We suggest that only the reserve of colloid is stored in the thyroid gland by the process of colloid storage.

We have already described elsewhere[18] the decades in the life of the individual at which secretion is the main, though never the sole, activity of the thyroid mechanism. In the embryo and the new-born, secretion seems to be the only product of the gland. It is well to remember that at this

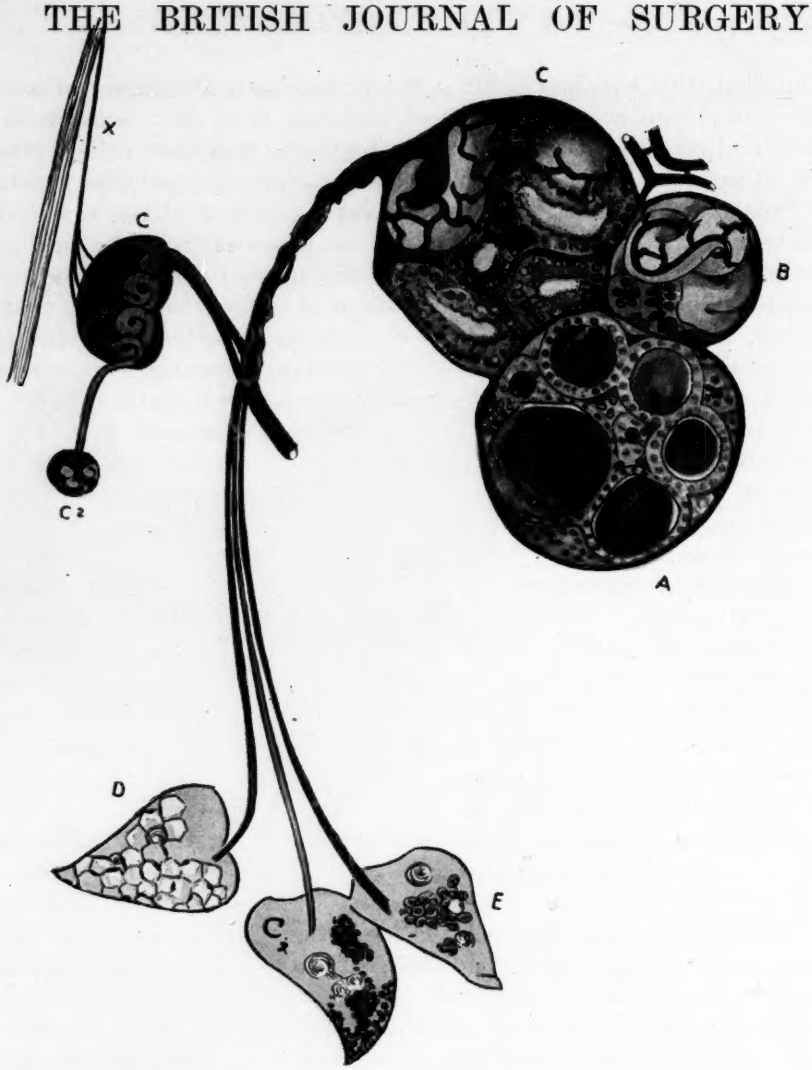

FIG. 311.—Diagrammatic summary of the histophysiology of the thyroid apparatus. *A*, A gland-unit engaged in accumulating and storing colloid by the process of vesiculation. This occurs in defined areas of the epithelial columns, and leads to fragmentation of the columns into tense discrete spheroidal vesicles. The ground tissue of the gland-unit appears as a pavement of epithelial cells, representing the roofs or floors of vesicles in tangential section. No demonstrable biochemical disturbance of the nuclei or protoplasm of the epithelium accompanies vesiculation. Such a gland-unit must arise from, or revert to *B*, the rest phase of the thyroid unit. This phase has long been recognized as the loosely termed 'fœtal rest tissue'. It does resemble embryonic thyroid tissue, but it is the common state of the thyroid in starvation and marasmus, and may occupy the whole gland in some normal individuals whose turnover is at a low level. The nuclei are orientated in an indefinite manner about the central intra-epithelial microcapillaries, in contrast to the regular pavement arrangement in the colloid unit. This rest phase always intervenes between the colloid stage and *C*, the secretory phase. The sequence of events in the transition from rest to activity seems to be as follows. The sinusoid opens and contains a fluid and lymphocytes; the nuclei move to the sinusoidal surface of the epithelial column; the protoplasm becomes granular and later vacuolated; the vacuolar fluids flow to the central zone, forming a lake (the process of lacunation); there is no tension in the cavity so formed. The microcapillaries fill with fluid and granules. Secretion so formed is not stored in the follicles, but appears in the lymphatics leading from the sinusoid; in the parathyroid (C^1), where it gains intimate contact with the sympathetic nervous system X; and in the lymphatics leading to the thymus, in which it induces a lymphoid phase. The thymus can be traced through three phases: a vesiculated fat (D), a granular lipoid (E), and a lymphocytic activity (C^2). It is only in the secretory phase that the co-operation of thyroid, parathyroid, and thymus is evident.

age the gland is devoid of iodine. After birth colloid storage increases up to five years of age, while secretion declines. From nine to fifteen years the position is reversed; secretion predominates in the work of the gland, and may overshadow the colloid-storage function. Thereafter up to eighteen to twenty years, sex determines the picture; the female presents in the main a secretory picture, and the male in the main one of colloid storage. After that age the female approximates to the male type, except in pregnancy and perhaps in menstruation. In his recent book, de Quervain, speaking of secretory activity as 'hyperplasia', seems to confirm this distribution of the alternation of the two processes in the gland at different ages.

The above summarizes the condition in the average individual. It must be remembered, however, that this average covers very widely separated extremes. Every individual seems to be a law to himself in the demands made upon the secretory activity of his thyroid apparatus; a physiological enlargement may be a necessity. We would remind our readers that the liver, the kidneys, and the lungs—in fact, all the organs—conform to this last rule. It is perhaps fortunate that swellings in these organs are less obvious than those of the thyroid gland, and that it is thus less possible for the uninitiated to measure their varying morphology in pathological terms.

Before passing on to the clinical application of our work, it is necessary to emphasize the fact that any excess of secretion of the thyroid gland is stored mainly outside the thyroid, in the thymus, and it may be in all lymph-endothelial tissue. Normally, at the height of the secreting process, slight accumulations of secretion are found in the centre of the epithelial columns (lacunation of secretion). Any delay in the evacuation of this secretion from the follicles is unusual in the normal gland. Indeed, the whole pathology of secretory activity seems to turn upon the failure of the tissues to effect a natural re-absorption of the secretion. We would point out that, according to modern views, a similar failure on the part of kidney parenchyma to re-absorb is the basic factor in the pathology of nephritis.

For the clinician the important points to remember are (*Fig.* 311): (1) That secretion is not colloid; (2) That intra-glandular storage of secretion is rare in the normal gland; (3) That colloid is a separate entity; (4) That there is an essential need to maintain in the gland a reserve of colloid which on demand is free to circulate in the body generally.

It is possible now for clinical purposes to trace, in the exaggerations and disorders of these functions, the significance of certain common forms of goitre.

APPLIED PATHOLOGY.*

A detailed study of the pure pathology of the goitrous thyroid gland has already been published by us[17], and should be consulted for a fuller understanding of the morbid physiology and anatomy underlying the various goitres. It is characteristic of the 'visceropathies'[19] in general that the associated symptom-complex is an expression of the failure of the

* The following sections form part of a thesis presented for the M.D. Edinburgh, and awarded a gold medal.

compensatory mechanism. One symptom-complex may thus follow a variety of pathological conditions (e.g., in renal and cardiac disease). This also appears to be the case in the diseases of the thyroid gland. Because of this, it is seldom possible to present a satisfactory classification of the clinical conditions if the clinical nomenclature of diagnosis is a mixture of 'pure' and 'applied' pathology. When a simple constant morbid state underlies a constant clinical syndrome, pure and applied pathology may coincide; but even under these conditions it is not safe to assume that the pathological condition in the organ is the etiological factor in the production of the symptoms—i.e., that one is the cause and the other effect. This is, of course, well recognized in the diseases associated with renal disturbances, but is less well recognized with the so-called endocrine glands. The fact that insulin exercises a profound influence in diabetes has not established the pancreas as the primary and only seat of the disease of which diabetes is the symptom. Any of the many 'thresholds', as the physiologists term them, may be the seat of the damage—the threshold of 'absorption', of 'co-ordination', of 'utilization', or of 'excretion'. We are as yet in ignorance of the body function over which the thyroid apparatus presides, so that we know nothing of various 'thresholds' of activity and exchange concerned. Without this knowledge we cannot hope to reach a satisfactory understanding of the etiology of the diseases associated with goitre. Indeed, we cannot even approach the problem until we recognize the foregoing fact, and cease to be content with the vague satisfaction provided by the 'endocrine' theories.

DISORDERS OF SECRETION.

Two disorders of secretion demand particular consideration: (1) *Graves' disease;* (2) *Simple thyrotoxicosis.*

1. **Primary Graves' Disease.**—Graves' disease is associated with many pathological types of goitre. Berry,[20, 21] Plummer, Wilson, and Boothby[22] have already shown that Graves' disease proper has a distinctive pathology.

We have recently demonstrated that there is one type of goitre in which this specific histopathology occupies the *whole* of the substance of the thyroid gland. Such a goitre we have termed 'adenoid goitre'.[17] Clinically, adenoid goitre has a 100 per cent coincidence with the full syndrome of Graves' disease. Pathologically, adenoid goitre has a second 100 per cent coincidence with the *absence of colloid vesicles* in the gland.

If we look at the histology of adenoid goitre, three distinct and easily recognizable stages appear in its progress. In the first stage the tissue is uniformly in a state of secretory activity, while the physiological factor of vesiculated colloid (i.e., colloid storage) is absent. According to our studies of the normal physiology of secretion, it is essential for the balanced action of the gland that colloid should circulate through the parenchyma. It seems natural to conclude that the absence of any reserve of colloid would inhibit the proper production of secretion: thus secretion produced in the absence of colloid cannot exercise its normal effect.* The action of such a secretion

* This defect, as Boothby suggests, may be due to a lack of available iodine with which we know the colloid to be associated.

upon the body is unphysiological. The disturbance from a histopathological point of view is a secretory dysthyroidism—a conclusion which has already been reached by many observers on purely clinical grounds.

There is, moreover, another point of importance in the light of our conception of the thyro-thymic mechanism. Post-mortem examination of cases of adenoid goitre reveals the fact that, although the thymus is enlarged, at no stage does it contain an excess of lymphocytes. It is, on the contrary, in the endothelioid phase of its physiology. The thymic reservoir presumably, as indicated by the absence of the lymphocytes, is not being called upon to store any excess of the secretion that is so obviously being produced in the thyroid gland. This absence of lymphoid hypertrophy in the thymus has also been commented upon in particular by Gebele,[23] Halsted,[24] Crotti,[25] and Falta,[26] and is still further emphasized by many Continental observers, who separate from true Graves' disease a 'thymic Basedowii' which has its own peculiar pathology. The significance of this abnormal participation of the enlarged thymus in the inordinate activity of the thyroid in Graves' disease with an adenoid goitre would seem to point to the probability that all the viciously produced secretion is being utilized as it is produced. In Graves' disease associated with adenoid goitre histopathology indicates that both demand and supply are exalted to a pathological degree. This would imply that the cause of adenoid goitre lies in some factor extrinsic to the thyroid gland.

The next stage in the progress of adenoid goitre is the filling up of the follicles until, under the microscope, they are seen to be choked with secretion. When this occurs the sinusoids become empty, and few if any lymphocytes are found in the gland. This choking of the follicles probably means the forcing of secretion through unusual channels: for example, the blood-channels. It is perhaps analogous to the jaundice which follows choking of the bile-passages in the liver, or to the uræmic state which follows the choking of the kidney in parenchymatous nephritis. Clinically this second stage is signalized by the exacerbations of acute symptoms which occur in the course of the disease, and which may decline with treatment by rest. That is to say, the exacerbations represent, according to our view, the addition to the initial exophthalmic syndrome of a thyrotoxicosis, which is the result of the regurgitation of secretion from choked follicles, perhaps into the blood instead of into the lymph.

The third stage in adenoid goitre begins when a diffuse fibrosis of the walls of the sinusoids circumscribes the follicles as an impermeable membrane. The blood capillaries, however, lie between the fibrous wall and the 'naked' epithelium. This renders evacuation of secretion impossible through normal channels; the choking of the gland-unit becomes irreversible. The thyrotoxicosis then becomes continuous, and clinically the disease loses its exacerbative features. This third stage is the one in which partial extirpation of the gland is found to be satisfactory. The explanation is that the operation removes a mass of gland substance which contains, pent up within its fibrotic follicles, the perverted secretion which is toxic in its action. In this stage surgery is performing more quickly what nature is slowly accomplishing by atrophy and exhaustion of the epithelium.

Thus the pathology of adenoid goitre contains for the clinician the suggestion that primary Graves' disease presents the following sequence: (1) An 'exophthalmic' stage; (2) A superadded exacerbative thyrotoxic stage; (3) A more or less continuous thyrotoxic stage proceeding to atrophy and exhaustion. Each of these stages has its specific histology.

Such a sequence of events associated with a typical adenoid goitre we would term primary Graves' disease—primary, in that the cause is extrinsic to the gland.

2. **Simple Thyrotoxicosis.***—There is a thyrotoxicosis uncomplicated by exophthalmos. We think the term thyrotoxicosis (Kocher)[27] is preferable to such terms as 'forme fruste', 'Graves' disease without exophthalmos', 'toxic adenoma', etc., inasmuch as there is a specific pathology underlying the condition wherever it occurs. The condition may follow any of the many pathological changes which give rise to goitre. It has already been discussed as a specific part of the pathology and clinical syndrome of primary Graves' disease.

In the thyroid gland which is the seat of a progressive fine sclerosis, atrophy and degeneration of the gland substance take place. In a varying percentage of these cases this loss of gland substance is countered by a compensatory hypertrophy of the healthy *residuum* of the gland. It is this residuum of functioning tissue that provides the pathological basis for the thyrotoxicosis, and not the primary lesion, which is merely destructive. The compensatory hypertrophy may be due to excess of supply of metabolites from which secretion is produced, or to an excess demand for secretion; neither is absolute; both are relative to the available healthy gland tissue.

If attention is focused upon this residuum of gland substance, it will be seen that the continual progress of secretory activity may ultimately lead to the choking of the follicles with secretion; that is to say, by distention of the follicles the sinusoidal space is obliterated. On the assumption that was made for primary Graves' disease, we look to the regurgitation of this accumulated secretion as the cause of the thyrotoxic symptoms. The fundamental histopathology of simple thyrotoxicosis is thus the same as that found in the second stage of primary Graves' disease, viz., the accumulation of secretion distending and choking the follicles. In primary Graves' disease, however, the condition is general throughout the gland, whereas in simple thyrotoxicosis it is a focal one.

There is a difference as well as a similarity between these two conditions, for it must be noted that in simple thyrotoxicosis *colloid does not disappear* from the gland as a whole. It appears therefore that the overworking of the residuum of the tissue can lead to a choking of the follicles with secretion, and to a vicious resorption of the contents even in the presence of a sufficiency of colloid. Clinically the condition is associated with intoxication, and generally with goitre. The condition, more often than not, stops short at the thyrotoxicosis, constituting the so-called 'toxic adenomatosis' of Plummer[28] or the 'interfollicular adenomatosis' of Goetch.[29]

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* The word toxic is used here in a general rather than in the specific sense of the bacteriologist and pharmacologist.

Obstruction to the normal outflow of any excess of secretion is the fundamental pathology of simple thyrotoxicosis. On this basis a mechanical obstruction may occasion this result, and it may be that a simple tumefaction, by its bulk or its disposition within the gland, can result in simple thyrotoxicosis—toxic adenoma, or, as it would more accurately be described, adenoma complicated by thyrotoxicosis.

Secondary Graves' Disease.—Secondary Graves' disease is an immediate or remote sequel of a simple thyrotoxicosis.

The basic pathology of secondary Graves' disease is the same as that of simple thyrotoxicosis, in that it is the result of a compensatory hypertrophy in the functioning residuum of a gland which is the seat of some diffuse destructive lesion. Thus, like simple thyrotoxicosis, it represents a condition of affairs in which the effective or normal production of secretion is interrupted. In the course of the progressive condition, the hypertrophic residuum of tissue itself suffers from a fibrosis. This fibrosis is essentially of a different order from the primary lesion in the gland. It is the result of the overworking of the functioning residuum of thyroid tissue, and is of the order of 'strain fibrosis' described by Adami.[19] It is of the intimate variety which penetrates into the gland-unit and insinuates its fibres between the epithelial and endothelial surfaces of that unit. This results in the production of a fibrotic wall about the follicles. The strain of overwork has been operating on the whole residuum; colloid-storage areas and secreting areas alike are obstructed, so that colloid also becomes pent up within the fibrotic vesicles of the residuum of tissue. Colloid, therefore, though obviously present in the gland, *is now rendered unavailable for the purpose of function and circulation.* We return thus, but by a different route, to the condition of affairs that we have demonstrated in primary Graves' disease—namely, the presence of pent-up secretion within the epithelial columns, and the absence of circulating colloid. The difference between the primary and secondary Graves' diseases is that in one the histopathological condition in the gland is general, while in the other it is focal.

Naturally in all cases clear lines of demarcation between the individual events in the sequence which leads to secondary Graves' disease cannot be seen. For example, in secondary Graves' disease of the young, an early 'puberty goitre' can induce a perifollicular fibrosis. We have seen that the physiological demand for secretion in the female at this age is considerable. The residuum of the gland may therefore be compelled to undergo an extremely rapid hypertrophy, so that the preliminary thyrotoxicosis is masked by the rapidity of the progress of the disease. As a contrast to this, the secondary Graves' disease of the senile period (e.g., menopause) usually has years of simple thyrotoxicosis before exophthalmos ensues.

In a well-marked case of secondary Graves' disease this sequence of events is in marked contrast to that of primary Graves' disease, in which the exophthalmos occurs conspicuously early, and toxic symptoms follow later.

Pathology suggests that the main features of secondary Graves' disease are: (1) A preliminary exacerbative thyrotoxicosis; (2) A chronic, or

continuous thyrotoxicosis; (3) The supervention, at a later date, o exophthalmos to complete the syndrome. This is the inverse sequence to that of primary Graves' disease.

Discussion.—The pathology of function of the thyroid gland is identical in Graves' disease, whether this be of the primary or secondary variety, and wherever it be found in goitrous conditions.[17]

If the morphological appearances of the thyroid tissue in the two diseases be examined, this identity is not apparent. In the gland of primary Graves' disease (adenoid goitre) no colloid can be seen in the tissue, while in secondary Graves' disease (intralobular fibrotic goitre) varying amounts of vesiculated colloid can always be found, but each vesicle is surrounded by a fibrotic wall which renders the colloid of no avail from the point of view of function. Thus in both types of Graves' disease the disorders of function in the gland are the same, and consist in a perversion of secretory activity together with an absence of colloid capable of circulation through the gland. In neither case is the disorder of the nature of a ' hyperthyroidism '.

From our observations of the pathology of the two conditions, we are driven to the conclusion that the essential difference between primary and secondary Graves' disease is to be found in a critical examination of the *relation of the demand made upon thyroid activity to the capacity for response in that tissue.* In primary Graves' disease we must postulate an initial inordinate demand made by the body generally upon a normal and willing thyroid gland, while in secondary Graves' disease the condition is one of a normal demand made upon a previously injured thyroid gland. From what direction the insatiable demand upon the thyroid in primary Graves' disease comes we are quite unable to state, and can but suggest that the demand arises in some metabolic disorder analogous to, and as intricate as, that which underlies the disorder of carbohydrate metabolism in diabetes. We would suggest that this possibility in itself should be sufficient to cause the physician to return into the field of thyroid disease, and renew his attack upon its early manifestations.

It is to be noted that the lesion in secondary Graves' disease does not preclude the possibility of a sufficiency of colloid somewhere in the body, though this has not yet been demonstrated. It may be that iodine meta bolism is still possible outside the thyroid gland, as Boothby suggests.

With regard to the underlying cause in the thyroid gland which ulti mately makes the onset of secondary Graves' disease possible, we are equally uninformed. The pathology of the condition is analogous to that of chronic interstitial nephritis. Thus, in this branch of thyroid disease also, it is to the clinician that we must look for an elucidation of the earlier factors in the etiology of the disease.

In these conclusions, based upon histopathology, lies a satisfactory sub-stantiation of the work of the Mayo Clinic. Plummer, Wilson, and Boothby seemed to have reached a similar, if not identical, differentiation of the types of Graves' disease from the clinical and pharmacological point of view.

It will be seen that in all these conditions the basic pathology of Graves' disease and thyrotoxicosis implies a degree, it may be a considerable one,

of *hypothyroidism*[17] (not hyperthyroidism) in the presence of the *dysthyroidism*.[30] [31] That is to say, there is an *insufficiency* as well as an *inefficiency* of the secretion.

Goitrous Tachycardia.—Thyrotoxicosis we have seen to be a factor common to both types of Graves' disease, and to be a clinical condition of itself. At this point it is as well to insist that the tachycardia that arises in many forms of chronic goitre (e.g., interstitial thyroiditis) is not necessarily the result of a thyrotoxication; nor is the anxiety and agitation secondary to the tachycardia at all related to the psychosis and tremor of true thyrotoxicosis. The workers of the Mayo Clinic emphasize this fact, and seek an explanation in some form of irritation of the sympathetic nervous system. If we recall the heavy sympathetic innervation of the thyroid gland, it seems not at all improbable that a simple interstitial fibrosis may irritate the nerves, or that retrogressive changes which occur in the gland substance may also involve the intimate nerve-fibres, inducing a form of neuritis, apart altogether from any thyrotoxicosis.

Summary of Disorders of Secretion. — Primary Graves' disease, secondary Graves' disease, and simple thyrotoxicosis have certain histopathological features in common, just as they have certain clinical features in common.

Primary Graves' Disease.—Primary Graves' disease affects the whole gland, producing an adenoid goitre. Some extrinsic cause for the change in the gland seems certain. The stimulus to secretion is overwhelming, absolute, and not relative. Colloid disappears from the gland, and later becomes insufficient to serve the body function. In the beginning the body completely utilizes the excess of viciously produced secretion. Later the body fails to utilize it; it becomes pent up in the follicles, and the excess overflows, to exercise a disturbing 'toxic' action on the tissues.

Thyrotoxicosis.—Thyrotoxicosis is always secondary to some gross primary lesion which reduces the functioning power of the gland. The stimulus to secretion is only relatively increased; loss of thyroid substance is the cause of this relative increase. Colloid in the glands, and perhaps also in the body, still remains sufficient to serve the body function. The amount of secretion becomes greater than the gland mechanism can eliminate. It is pent up in the follicles and overflows as a disturbing 'toxic' substance.

Secondary Graves' Disease.—Here also the stimulus to secretion is only relatively increased owing to the loss of gland substance due to previous injury. But in this case there is also an interference with the efficient circulation of colloid through the gland. The secretion is similarly shut up in the follicles, and again it overflows to exercise a disturbing 'toxic' effect.

DISORDERS INVOLVING COLLOID STORAGE.

Acute Vesicular Goitre.[17]—In studying the applied pathology of adenoid goitre, we noted the entire absence of colloid from the gland, and constant association with the symptoms of Graves' disease.

Acute vesicular goitre or colloid goitre has the inverse picture, an entire absence of secretory activity and the entire absence of subjective symptoms.* In these facts alone there seems to us to be convincing proof of the duality of functions in the thyroid gland. To credit colloid with the properties of a secretion, as evinced in Graves' disease, and at the same time to term the huge acute swelling (vesicular goitre), in which we know there is a vast increase in the actual amount of colloid, a sign of hypothyroidism, seems to be a contradiction in facts as well as in terms. This peculiar pathological condition, acute colloid goitre, is associated with a peculiar negative syndrome. Berry[32] was the first to note that acute colloid goitre is associated with a high incidence in the young male. Since we have been able to exclude from the group of colloid goitres the minor degrees of hypertrophic goitre (the old adeno-parenchymatous goitre—*see* p. 493), we find the incidence of acute colloid goitre in the young male almost as striking as the incidence of primary Graves' disease in the young female. The surgeon is familiar with this comparatively rare acute puberty goitre, since it is usually presented to him as an emergency matter—acute dyspnœa—presumably arising from the impaction of the descending swelling in the bony ring of the thorax. (Chronic goitres, of course, can be responsible for a compression dyspnœa.) It has been objected that the female escapes operation, and in that way the disproportion of incidence between the sexes arises. This explanation seems to us quite untenable. There is no anatomical reason in the female why an acute thyroid swelling should not descend and cause acute compression and dyspnœa. Indeed, from the high incidence of all goitres in the female, we would anticipate the reverse, unless, as we suggest, the acute colloid goitre is of greater general frequency in the young male at puberty as a specific condition. There can be no doubt that in this colloid enlargement, in its acute forms, the colloid material is pouring into the follicles of the gland. If colloid were produced in the manner of any secretion, the epithelium must manifest the extraordinary changes in form and content associated in other glands with the elaboration of secretion. Yet nothing looks so placid and passive as the epithelium; likewise, the sinusoids are empty, the lymph channels are not in evidence, and the endothelium of the distended capillaries is in a quiescent state. Everything, in fact, makes it clear that we are dealing with a peculiar and specific function.

We are thus drawn to the important conclusion that acute vesicular goitre has its own specific pathology and peculiar negative syndrome, and a high incidence in the male at puberty.

Special attention should be directed to this form of goitre from an experimental point of view, in that it is the antithesis of the adenoid goitre of primary Graves' disease. We see again, in these two conditions, that particular insistence on sex and puberty as an etiological factor to which little attention has been given in the experimental studies lavished on the thyroid apparatus. Gaskell's[33] phylogenetic studies of the thyroid gland are the only significant literature on this probable aspect of the thyroid function.

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* Excepting, of course, occasionally pressure symptoms which are mechanical.

Chronic Vesicular Goitre.—The thyroid gland in this form of goitre has the appearance of a collection of colloid cysts surrounded by fibrous tissue. In this type of goitre, as in the acute type of vesicular goitre, the characteristic feature is the absence of any secretory tissue. *In the acute variety there is an active over-production of colloid, whereas in the chronic variety the accumulation of colloid appears to be produced by stagnation of the normal physiological turnover.* The incidence of chronic vesicular goitre in the sexes is not disproportionate when judged by operation material. How far this incidence is due to the fact that chronic vesicular goitre is also one of the types of endemic goitre is difficult to say, until such time as the survey of endemic goitre has been made from a pathological point of view.

Chronic colloid goitre is only one variety of endemic goitre. It presents a very different picture from the other form of endemic goitre to be dealt with later, and probably shows an independent etiology. We will return to a consideration of chronic colloid goitre after describing the second variety of endemic goitre.

DISORDERS INVOLVING BOTH COLLOID AND SECRETION.

We have dealt so far with the two extremes, in which either colloid is sacrificed to secretion, as in Graves' disease, or in which secretion is sacrificed to colloid, as in vesicular goitre. Clearly there must be a state of affairs in the thyroid gland wherein both these functions are exaggerated, constituting a true balanced hypertrophy.

It should be noted that hypertrophy is always a compensatory effort, and that the rule must hold that only the sequelæ of over-activity will lead to symptoms other than mechanical. In considering hypertrophic goitre it is sufficient to mention the physiological goitre of childhood, pregnancy, menstruation, and those of iodine medication and thyroid feeding. These physiological and pharmacological goitres are acquiring great significance because of the tendency to look upon all rises in the basal metabolic rate as indicative of pathological thyroid upset. The raised basal metabolic rate is, however, only another form of fever—apyrexial fever—and, as such, should take its place in diagnostics as indicating a necessity for compensatory effort somewhere in the metabolic cycle, but not necessarily in the thyroid, nor indeed necessarily pathological.

Chronic Hypertrophic Goitre.—We may turn to the consideration of one of the most important forms of hypertrophy in the thyroid gland, that of *chronic hypertrophic goitre.* This goitre is a true hypertrophy; overwork induces strain, and this results, as is usual, in a diffuse fibrosis throughout the gland. This fibrosis circumscribes areas of secretory hypertrophy (solid adenomatous tissue) and areas of excessive colloid storage (colloid cysts). In this country it is commonly called 'adeno-parenchymatous' goitre, and is one of the commonest forms of thyroid swelling.

Apart from its sporadic incidence, the importance of this goitre is that it constitutes the second variety of endemic goitres. It is undoubtedly the form of endemic goitre that was studied by McCarrison[5] in Gilgit, and it

coincides with one type of goitre prevalent in Switzerland and described by de Quervain[34] in that country. The etiology of the hypertrophy is still unknown.

ENDEMIC GOITRE.

It would appear from our studies that in the *adult* there are two varieties of goitre found endemically. In our opinion, one of the most valuable results of recognizing the existence of secretion as something apart from colloid-storage in the thyroid physiology is that it enables us to distinguish a chronic vesicular goitre from the chronic hypertrophic forms of goitre.

De Quervain (*see* Aschoff[35] also) in his recent work illustrates the two types of endemic goitre in patients of all ages. One type is undoubtedly the chronic vesicular goitre; the other variety, which he terms 'hyperplastic', corresponds histologically to our chronic hypertrophic goitre. He, however, draws no physiological distinction, as we do, between these two forms. The moment a physiological distinction is made, we have at hand a probable explanation of the very diverse results in the treatment of endemic goitre, and an explanation of the contradiction concerning the etiology of endemic goitre. It is improbable that the etiology of these two goitres can be the same; the correct treatment in each case will probably vary with the etiology.

There is a vogue for the indiscriminate treatment of endemic goitre with iodine. This is the revival of an old empiricism which no doubt lapsed for some good reason. The researches of Marine are largely responsible for the revival of iodine treatment. Marine maintains that iodine converts a 'hypertrophic' gland into a 'colloid' gland.[31] Chronic vesicular goitre, nevertheless, is undoubtedly the goitre that 'softens and dissolves', according to Kocher, under iodine medication. If Marine's contentions were correct, chronic vesicular goitre could only be aggravated by iodine therapy. McCarrison,[5] dealing with the chronic hypertrophic variety, finds the therapeutic administration of iodine useless. Indeed, in some zones, iodine appears to be a dangerous measure, as the figures of Fleischmann indicate.[36] He finds iodine may be toxic, and induce a so-called iodine-Basedowii (in Basel in 68 per cent of cases, in Berne in 28 per cent of cases, in Berlin in 37 per cent of cases). In Heidelberg, Krehl[37] finds iodine dangerous, and such authorities as Breuer, Möbius, and Ortner find a class of endemic goitre in which iodine is contra-indicated. (For a summary of these and other facts concerning iodine medication, *see* Falta.[26]) Whatever may be the effect of iodine in the goitre of children, we have said enough to indicate a need for the differentiation of endemic goitres before engaging in any specific treatment.

The basis of a differentiation appears to us to lie in the fact that vesicular goitre is functionally and etiologically different from goitrous swellings due to secretory hypertrophy (adeno-parenchymatous goitre), and that puberty and sex are important factors in their development.

In any case, it is a step forward to recognize *that there are two types of goitre with two distinct pathological expressions in endemic zones*, and, further, to recognize that these may be differentiated by the zone, or possibly by the sex in any one zone. In these considerations lie the grounds for a renewed attack on the problem of the etiology of endemic goitre.

CONCLUSIONS.

I have summarized our particular conclusions at the end of each section. There remains only to mention the more general conclusions we may draw from this study.

Early Graves' disease and early acute vesicular goitre are apparently diseases of that function of the body over which the thyroid presides, and not, primarily, of the thyroid gland itself; whereas secondary Graves' disease, simple thyrotoxicosis, chronic vesicular goitre, and chronic hypertrophic goitre are primarily affections of the gland itself, and only incidentally and occasionally affect the functions of the body over which the gland presides.

It is necessary to seek out the precise function over which the thyroid apparatus presides if we are to unravel the complicated tangle of the thyroid problem. Had insulin (the endocrine secretion) been the first discovery in the study of the function of the pancreas, the precise position of the pancreas in relation to digestion might still be awaiting elucidation. We suggest there is a major function as well as an 'endocrine' one for the thyroid gland.

All that has been attempted in this paper is to present this problem anew to the clinician, and to indicate to him the various directions in which pathology points towards its solution.

We are indebted to Sir James Berry for the opportunity his material has afforded for this research work, and to him personally for his continued encouragement and help in the work. We have pleasure in acknowledging our gratitude to the Council of the Royal College of Surgeons for the opportunity of repeating many of our dissections in their laboratories, where the pervasive Hunterian atmosphere, and the help and encouraging criticism of Sir Arthur Keith, make labour light. We thank Dr. Hilda Cunnington for her continued help in the preparation of material.

Mr. Sewell's drawings speak for his patience and skill, and are an index of our considerable obligation to him.

This work is the substance of a report presented to the Medical Research Council. Personal association with Sir Walter Fletcher has been a real stimulus to work.

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BONE-GRAFTING OF THE MANDIBLE: WITH REPORT OF SEVEN CASES.

By WILLIAM BILLINGTON and HAROLD ROUND, Birmingham.

For three years the writers, in association with Mr. Arthur H. Parrott, M.D.S., L.D.S., were in charge of the 'Jaw Centre' at the 1st Southern General Hospital, Birmingham, to which all cases of injuries to the jaws requiring special treatment were sent from the whole Southern Command. Upwards of 2000 soldiers were admitted to the Centre and treated by us. A very large percentage of these suffered from compound fractures of the mandible. Successful treatment involved: (1) Osseous union; (2) Functional occlusion; and (3) Avoidance of disfigurement.

In a number of cases the fracture of the mandible was complicated by considerable loss of bone, either occurring at the time the wound was received or subsequently from necrosis, and a considerable gap existed between the fragments. It was found that if the gap exceeded half an inch, osseous union was rarely obtained unless (1) the fragments were allowed to approximate at the expense of normal alinement, or (2) a bone-graft was successfully introduced. In a number of cases the loss of bone was so extensive that any attempt to obtain union by approximation was impracticable. The need for a satisfactory method of securing union by means of grafts was very great, and much experimental work was carried out before a technique was arrived at which could be trusted to give uniformly good results. A description of the method adopted was published in the *British Medical Journal*, Dec. 21, 1918, and in the *Proceedings of the Royal Society of Medicine*, 1919, vol. xii (Section of Odontology, pp. 55 to 72). Altogether upwards of 75 cases of compound fracture of the mandible were successfully treated by means of bone-grafts, the gap varying in extent from half an inch to five inches. The longest graft employed was seven inches in length; it was successfully used in the case of a soldier whose lower jaw from angle to angle had been completely destroyed.

The knowledge gained by our experience of jaw-grafting in connection with war injuries has enabled us to remedy successfully the deformities and functional disabilities resulting from loss of a portion of the mandible as a result of the accidents and diseases of civil life.

The conditions for securing successful jaw-grafting are better in civil practice than they were in connection with war injuries. In the latter class of case we had to contend with extensive associated damage to soft tissues, and with virulent and prolonged sepsis, from which dense scars of low vitality resulted. These formed a very unsatisfactory bed into which to introduce the graft. As the result of our experience, we feel justified in claiming that, provided there is no active disease present, defects in the lower jaw can be

successfully remedied by bone-grafts, and firm osseous union obtained without loss of normal alinement in practically every case. The length of the gap between the fragments is immaterial. The technique is simple, but does involve close collaboration between surgeon and dental surgeon.

The mandible is peculiar in that success in grafting can rarely be obtained by the technique used in connection with the long bones. It is very intolerant of foreign bodies of any kind, and attempts to fix a free graft by wire, pegs, or plates almost invariably led to failure. Equally, fixation by dove-tailing the graft between the fragments was frequently unsuccessful. Further, fixation of the fragments by dental splints led to absorption of the graft if carried out before the operation or until the wound had firmly healed. After many experiments, all attempts to fix the graft or the fragments were abandoned, fixation only being secured by dental splints about a fortnight after the graft was introduced and the wound firmly healed.

Pedicle grafts have a very limited application, and are only of value when the gap is a comparatively small one. A further disadvantage entailed by their use is that the fragment from which the graft is taken is weakened and greater interference with the teeth is necessary. Owing to the ease with which free grafts can be introduced, and the uniformity of success that has attended their use, the employment of pedicle grafts has been entirely discontinued.

A matter of considerable importance is the choice of the bone from which to take the graft. Rib bone is too soft, and does not develop strength equal to that of the jaw generally. Grafts from the tibia are brittle, and cannot readily be bevelled and shaped to fit the gap; also, tibial grafts do not appear to accommodate themselves to their new environment, and are apt to necrose and work out. After experimenting with grafts taken from various parts of the body, the choice finally made was the crest of the ilium. The anterior superior spine and the crest as far back as necessary is freed from the attached muscles, and a graft as long as required is readily obtained by sawing through the bone below the crest from the anterior superior spine backwards. The advantages are that the graft is readily obtained and a minimum of injury inflicted—in no case has any subsequent disability been complained of. The bone is very tough and can be cut to any required shape by bone forceps without splintering, and the graft is thick and strong. Finally, grafts obtained in this way readily accommodate themselves to their new environment, and their nutrition rarely causes any anxiety. It is quite exceptional for any indication of absorption or rarefaction to be seen on X-ray examination between the time the graft is introduced and its firm osseous union with the fragments.

OPERATIVE TECHNIQUE.

Preliminary Preparation.—It is absolutely essential that there should be no sepsis present in the field of operation. If this has existed, there should be an interval of several months after all signs of inflammation have subsided and the wound has firmly healed before a grafting operation is attempted. Unless this precaution is taken, there is risk of latent sepsis

lighting up. Great care should be taken to exclude the presence of sinuses opening into the mouth. Teeth immediately adjacent to the gap must be extracted, and all septic teeth or stumps in any part of the mouth removed. Just before the operation all splints and fixation apparatus should be removed, and no splints should be introduced for at least two weeks afterwards when the wound has healed. The presence of fixation splints within the mouth causes risk from post-anæsthetic vomiting, greatly adds to the discomfort of the patient, and, where pressure is exerted by them in or near the operation area, interferes with vitality and increases the risk of sepsis.

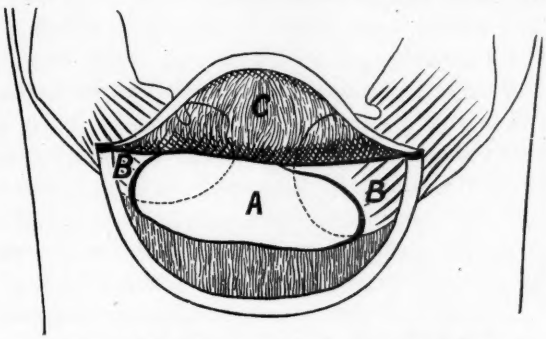

FIG. 312.—Diagram showing preparation of bed by raising a musculo-cutaneous flap (C). The graft (A) overlaps the bevelled ends of the jaw fragments (B B).

Operation.—A curved incision is made through the skin of the neck, beginning a full inch behind the extremity of the posterior fragment and ending a similar distance in front of the extremity of the anterior fragment (*Figs.* 312, 313). The incision commences and finishes about half an inch above the line of the lower border of the jaw and in the neck runs about an inch below that line. The incision is deepened by cutting upwards and inwards until the lower border of each fragment is reached. The soft tissues covering the outer surface of each fragment are then raised for an inch away from the gap and turned up in the flap. The ends of the fragments, together with the intervening fibrous tissue occupying the gap, are next carefully cut away. At this stage care is necessary to avoid making an opening into the mouth, an accident which necessitates abandonment of the operation and postponement until the wound has healed. The preparation of the bed is completed by removing a flake of bone from the outer surface of each fragment for an inch away from the gap. In doing this the exposed surfaces of raw bone are bevelled a little. All

FIG. 313.—To illustrate gap in mandible. The outer plate is removed from each fragment to the dotted line.

bleeding is carefully arrested, and the wound covered up while the graft is being prepared.

The graft is taken from the same side of the body as the wound in the face. This ensures that the patient will be able to lie comfortably on one side when sleeping. An incision is made over the crest of the ilium, commencing at the anterior superior spine and extending as far back as required. The muscles are then separated on either side of the crest by cutting through their attachments with a scalpel as close to the bone as possible. The soft tissues are pressed back by ribbon retractors, and the bone is made to project for nearly an inch. The graft is cut with an ordinary Horsley hand-saw with movable back. The division of bone commences in front below the anterior superior spine, and is carried back until a graft is obtained which is two inches longer than the gap in the jaw. It is advisable to measure the length required on the crest and to make a vertical saw-cut at the required spot before commencing the horizontal division. This ensures that when the saw has travelled the required distance the graft becomes loose.

The detached piece of bone is held in a sterile mop, its extremities are bevelled with bone forceps, and any adhering soft tissue is trimmed away with scissors. The graft is then introduced into its bed in such a way that the bevelled ends overlap the gap by an inch at either end and lie on the prepared raw surfaces on the fragments (*see* ***Fig.*** **312**). A little shaping is required to allow the graft to lie comfortably in place, but as the curve of the crest of the ilium approximates closely to that of the jaw, very little moulding is required. No attempt is made to secure fixation of the graft; it merely lies in the gap with its bevelled extremities extending over the fragments of jaw at either end. Two advantages result from the extensive overlapping: (1) A broad line of bony contact between the graft and the fragments is provided, with increased prospect of speedy, firm, osseous union; (2) There is practically no risk of separation in the event of the size of the gap being increased by the subsequent introduction of the dental fixation splints, as a certain amount of sliding can take place without contact being lost.

The soft tissues are sewn closely over the graft with chromicized catgut. This keeps it in place, has the additional advantage of surrounding the graft with living vascular tissue, and abolishes dead spaces in which blood-clot and serum can collect. Finally, the skin is approximated with a few interrupted stitches. No drainage is employed beyond that of leaving spaces between the skin sutures to allow of the escape of serum. A simple dressing and bandage is applied and the patient sent back to bed. The whole operation can be carried through deliberately and comfortably in about forty minutes. The patient complains of very little discomfort in the jaw, the chief complaint being directed to the wound in the hip.

No attempt is made to introduce the dental splints until the wound is firmly healed and the compound fracture has been converted into a simple one. This usually occurs in about two weeks, after which the case is treated as one of simple fracture of the jaw. Firm bony union occurs in from two to four months; but it is inadvisable to fit the final dentures until an interval of about six months from the operation.

Dental Technique after the Operation.—In from two to four weeks, according to the state of the wound and the patient, new models are taken and splints made for the fixation of the jaw in correct alinement. Any slight alteration in the occlusion, or any displacement of the fragments which may have resulted from the operation or in the interval prior to the re-adaptation of splints, is found to be easily reducible. This is possible owing to the fact that the graft, whilst being firmly held in position by the deep layer of soft tissues, overlaps the fragments at each end, and a certain amount of sliding can take place without bony contact being lost. The correction and retention of the fragments in their required positions is attained by the use of articulating splints, silver cap-splints adapted and cemented to any standing teeth, such splints being supplemented by vulcanite extensions. These extensions are lined with soft rubber where covering edentulous parts in the neighbourhood of the graft, in order that the parts may be retained in position with a minimum amount of pressure. The splints are fixed to each other by means of articulating tubes and bolts.

PARTICULARS OF CASES.

Case 1.—Master F. S., age 11. In March, 1919, while playing with another boy on his father's farm, he was wounded by the accidental discharge of a loaded sporting gun. The left side of the face was severely lacerated, and the mandible between the left lateral incisor and a point just in front of the angle on the same side was completely destroyed. A preliminary plastic operation on the cheek and lower lip was performed by Dr. McLeod of Shrewsbury, and the right fragment of the mandible was maintained in good occlusion with the maxilla by a vulcanite splint having an inclined plane on the right side which caused the right fragment, on closure, to bite into normal position. We saw the patient in consultation in June, 1920. The wounds were firmly healed, but a wide gap existed in the mandible. It was decided to close the gap by grafting as the only means of securing union without great sacrifice of normal alinement.

FIG. 314.—*Case* 1. Radiograph of jaw six years after insertion of graft (side view). The dotted lines indicate the graft. A few spicules of lead are still present.

Continuous metal cap-splints with articulating tubes were made for the maxilla and mandible. Considerable difficulty was experienced in their construction and fixation, both on account of the age of the patient and because there were very few teeth to use. The incisors in the maxilla had all been damaged, and the temporary teeth, most of which were carious and some loose, had to be used, the support obtainable from the permanent teeth not being sufficient.

In June, 1920, a grafting operation was performed. The bed was prepared in

the usual way, and the graft taken from the crest of the left ilium. Part of the graft consisted of cartilage. The graft 'healed in' without complications, and three weeks after the operation the fixation splints were inserted in the mouth. A soft vulcanite block, fixed to the lower splint by means of vertical pins secured by vertical tubes on the left side of the lower splint, was inserted. This block had a horizontal tube fixed to its upper surface which articulated with two horizontal tubes on the upper splint. A pin passed through the three tubes bolted the upper and lower splints together, and at the same time held the block in position. This appliance remained fixed until October, 1920, when the splint became loose and was removed, Union was then found to have taken place at both ends of the graft, but as it was very 'springy' the splints were refixed and not removed until December, 1920, when good firm union was established. This was six months after the insertion of the graft. *Fig.* 314 shows the jaw at the present time.

Case 2.—Mrs. B., age 34. In February, 1912, the late Mr. Jordan Lloyd excised two inches of the mandible for sarcoma. The patient was then 25 years old. Immediately after the operation one of us (H. R.) fixed the right fragment of the mandible into correct position with the upper teeth by means of interdental wiring, the upper and lower teeth being wired together. In four weeks the wires were removed and a restoration denture made. This and subsequent dentures were worn for nine years, when the patient was persuaded to submit to a bone-grafting operation.

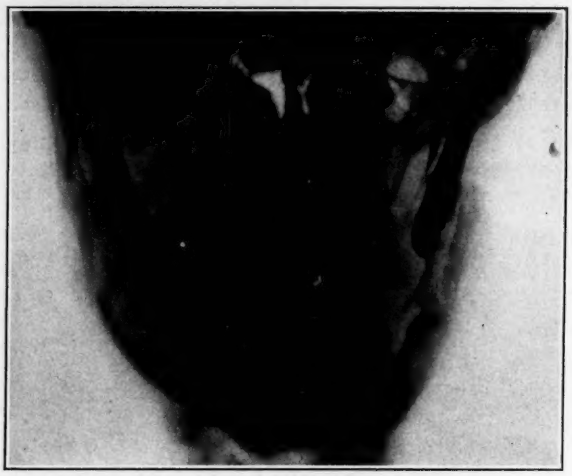

FIG. 315.—*Case* 2. Radiograph of jaw taken four years after insertion of graft.

In the interval between the two operations the patient had lost all her upper teeth, but she still had some teeth in the right fragment of the mandible: the left fragment was edentulous. The grafting operation took place in February, 1921, and two weeks later, when the wound had firmly healed, a vulcanite plate was made for the maxilla, and a metal cap-splint for the teeth on the right fragment. A movable soft vulcanite block attached to this articulated with the vulcanite upper splint on the left side. The under surface of this block fitted that area in the mouth corresponding to the position of the bone-graft and extending on to the posterior fragment, so helping to steady this and the graft. All parts were fixed together by tubes and pins. A chin splint made of aluminium, and struck up on a model of the patient's chin, was worn; it was kept in place by elastic webbing passing over the scalp. This splint was necessary for a short while, because the upper plate, whilst being fixed to the lower splint, could not be fixed to the maxilla. The wearing of the chin splint quickly accustoms the patient to keep the mouth closed, and when this habit has been acquired the splint can be dispensed with.

Firm union had taken place at both ends of the graft three months after the operation. As a precautionary measure a restoration denture was worn for a further four weeks, making four months in all. *Fig.* 315 shows the radiographic appearance four years after the operation.

Case 3 (double bone-graft).—Pensioner Albert W., age 30. This man was admitted into the Queen's Hospital with an ununited fracture of the mandible resulting from war injury. There was a gap in the region of the first and second molar teeth on the left side. A bone-graft was inserted on Feb. 17, 1921, and

fixation splints were inserted on March 9. Good union took place, and the splints were removed on July 13, 1921.

He went to the United States, and in December, 1923, was assaulted and robbed by a gang of roughs. He was hit on the right side of the jaw with a piece of lead and the right side of the mandible was fractured. He was admitted into hospital

FIG. 316.—*Case* 3. Radiograph showing first graft four and a half years after its insertion.

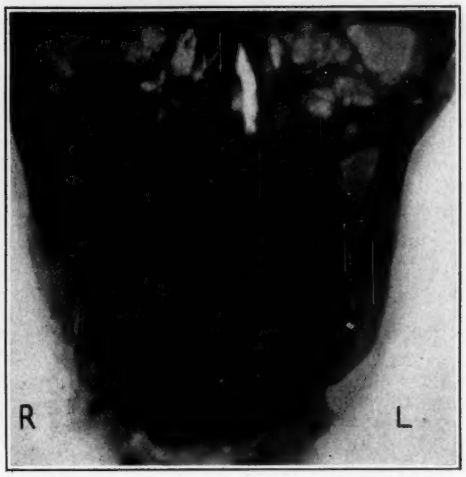

FIG. 317.—*Case* 3. Radiograph taken four and a half years after insertion of graft on left side and one year after insertion of graft on right side.

in Philadelphia, and the fragments were wired. This was followed by suppuration and necrosis, and he was sent home to England with a discharging sinus and the wire *in situ*.

He was again admitted into the Queen's Hospital on Feb. 24, 1924. The wire and several sequestra were removed, and fixation splints applied in the hope of obtaining union. The wound healed, but there was no attempt at union. On July 1 a bone-graft was inserted. This healed in without trouble, and firm bony union had taken place in two months. The original graft had become firmly welded into the jaw, from which it could with difficulty be distinguished (*Figs.* 316, 317).

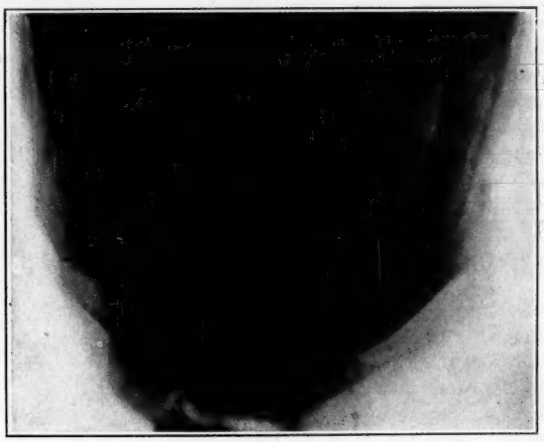

FIG. 318.—*Case* 4. Radiograph of jaw three years after insertion of graft.

Case 4.—C. Evans, age 23. About seventeen years ago, at the age of 6, a portion of the right side of the mandible was removed by Mr. Leedham-Green for sarcoma. The fragments were kept in fair position for some years by bridging the six-year-old molar on the right fragment to teeth on the left fragment. Later the bridge became useless and the parts approximated considerably. On April 5, 1922, a bone-graft was inserted, and good union resulted in four months. *Fig.* 318 shows the condition three years later.

Case 5.—Florence Parker, age 8. This child suffered from severe osteomyelitis of the mandible when 5 years old. Considerable necrosis occurred, and a gap in the continuity of the mandible resulted. The gap was situated just in front of the angle on the right side. A graft was inserted in April, 1922. The graft, which was composed largely of cartilage, was taken from the crest of the ilium. Some doubt was felt whether a graft mainly composed of unossified cartilage would be a success. This doubt was intensified by the fact that within a week of the insertion of the graft the child developed scarlet fever, and she had to be sent away to a fever hospital without fixation splints. These could not be applied for over a month. Firm union was, however, obtained four months after the operation. *Fig.* 319 shows the result after three and a quarter years.

FIG. 319.—*Case* 5. Radiograph of jaw three and a quarter years after insertion of graft. An unerupted tooth is shown in the posterior fragment.

Case 6.—Mrs. B. For twelve years this patient had suffered from attacks of neuralgia in the right side of the head. These attacks became increasingly worse. On X-ray examination, the second and third molars were found malplaced and unerupted. The teeth lay horizontally in the jaw in the region of the angle, with their crowns in juxtaposition and locked together. The roots of the third molar extended backwards into the ascending ramus, and those of the second molar downwards and forwards just in front of the angle. Both teeth were placed centrally to the jaw, and the surrounding bone was extremely thin and dense.

With considerable difficulty the teeth were removed in November, 1923, but the bone was so thin and brittle that a fracture occurred. The fragments were kept in as good alinement as possible by intermaxillary dental wiring until the wound had healed sufficiently to allow fixation of splints. The vitality of the bone at the site of the fracture was so poor that absorption took place.

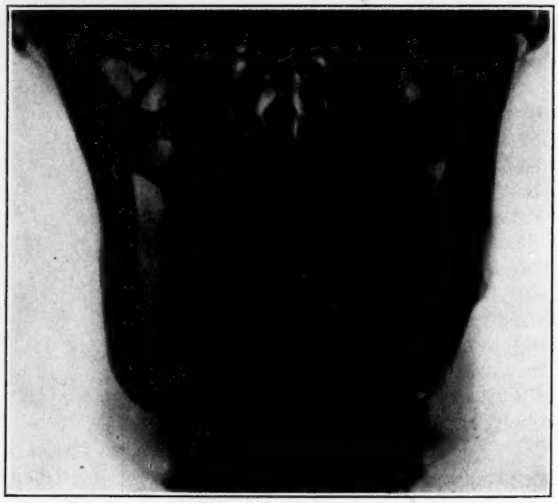

FIG. 320.—*Case* 6. Radiograph of jaw one and a quarter years after insertion of graft.

As there was no indication of union at the end of four months, it was decided to insert a bone-graft. The operation was performed on March 23, 1924. Uninterrupted recovery took place, and on July 5, 1924, firm union had occurred and the splints were removed. *Fig.* 320 shows the condition one and a quarter years after the operation.

Case 7.—Mrs. H. In January, 1924, the patient attended the Queen's Hospital complaining of pains in the right arm. For several months the pains were regarded as rheumatic, but as they became more severe in spite of treatment, the jaw was radiographed, and what appeared to be an odontome in the region of the second

bicuspid was shown. As the supposed odontome involved almost the entire depth of the jaw, it was decided to excise the section of the mandible in which it was situated. This was done, and it was then found that the trouble was caused by an unerupted second bicuspid which lay horizontally with its root incorporated in very dense bone. This obscured the root in the radiograph, the crown alone being

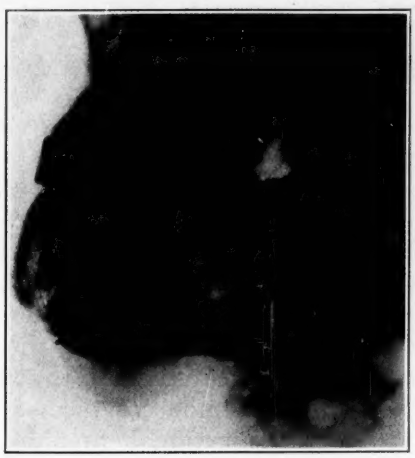

FIG. 321.—*Case* 7. Radiograph of jaw nine months after insertion of graft.

discernible. In September, 1924, the gap was closed by a bone-graft. Union took place rather slowly at the anterior end of the graft, but in February, 1925, it was satisfactory, and the splints were removed. The arm pains completely vanished after the operation in June. *Fig.* 321 shows a radiograph taken in June, 1925.

We desire to acknowledge the valuable help we have received from Dr. Brailsford in connection with the radiographs.

COMPLETE EXPOSURE OF THE RADIUS.*

By ARNOLD K. HENRY, Cairo.

The entire length of the radius is most easily exposed through an antero-lateral incision.

ANATOMY.

Three long muscles flank the bone upon its outer or lateral border—the brachioradialis, miscalled the supinator longus, and the long and short radial extensors of the wrist (*Fig.* 324). They can be retracted outwards when they have been relaxed by bending the elbow. These three muscles have origins which are proximal to the elbow-joint, and in order to mobilize them fully, the incision must extend proximal to the elbow. Further, this lateral group of three is strapped to the lower fourth of the radial shaft by two small muscles—which form an oblique prominence when the hand is prone. When the long muscles are raised from the bone these two short muscles are raised with them. The *pronator teres*, inserted at the middle of the radial shaft upon its outer face, will then be exposed, and full pronation of the hand will reveal an expanse of bone in the lower half of the shaft.

Fig. 322.—Anatomical relationships at the upper part of the radius. The white crescent between the two black insertions represents the bicipital bursa and shows how it lies in a bay formed by the supinator edge. The surgeon is guided to the bursa by the outer face of the biceps tendon.

The *supinator muscle* grasps the upper third of the shaft, and the *posterior interosseous nerve* (deep branch of the radial—B.N.A.) penetrates the anterolateral face of the supinator, and lies between two layers of the muscle: the deep layer separates the nerve from the bone. The supinator attachment to the radius skirts the edge of the bicipital tuberosity, which is covered in front by a bursa (*Fig.* 322).

The Deep Guide.—The edge of the supinator is obscured by the loose connective tissue that surrounds the recurrent radial vessels, but it can be reached with precision by following the outer face of the *biceps tendon*. The outer face of the tendon leads the surgeon to the bursa: the knife cuts through this to the tuberosity and at once affords the rugine a point of direct contact with the bone (*Fig.* 325). It is

* Shown at the Section of Surgery, Royal Academy of Medicine in Ireland, April 3, 1925.

then easy, starting at the edge of the muscle, to begin detaching the grasp of the supinator from the radius. The biceps tendon is thus the deep guide in this exposure, and the surgeon must keep strictly to its outer side or he may injure the ulnar vessels.

The recurrent radial vessels curve outward at the level of the bicipital tuberosity, and can be hooked up on the finger and divided. The finger then gains admission to the plane of cleavage between the pronator teres and the brachioradialis (*Fig.* 324). The anatomy of this plane favours the surgeon, for the radial artery runs within the sheaths of the pronator teres and the flexor longus pollicis, while the radial nerve (superficial branch of the radial—B.N.A.) is attached to the long lateral muscles which flank the radius. The separation of these neighbouring structures is thus made easy.

FIG. 323.—Incision for exposure of the radius. Divide skin and superficial fascia from the lower end of the biceps belly to the radial styloid. Begin dividing the deep fascia over the biceps tendon.

THE OPERATION.

Incision (*Fig.* 323).—With the patient's elbow extended and the hand supine, divide the skin and superficial fascia by a straight incision from a point at the outer edge of the biceps belly four finger-breadths proximal to the bend of the elbow, and terminating at the tip of the radial styloid. Divide and tie the large superficial vein which crosses the mid-third of the radius.

The Deep Guide.—First expose the biceps tendon by dividing the deep fascia at its outer side. Continue the division of the fascia throughout the wound with blunt-nosed scissors. Pass the

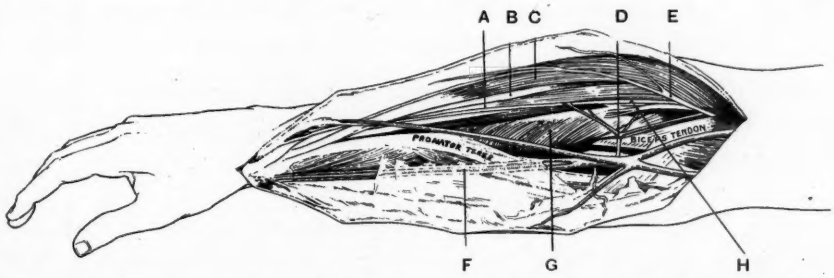

FIG. 324.—Exposure of the biceps tendon: slide a finger downwards along its *outer* side till it meets the resistance of the recurrent vascular loop. Divide and tie this loop. Mobilize the three long muscles which flank the radius: flex the elbow and retract them outwards. A, Extensor carpi radialis brevior; B, Extensor carpi radialis longior; C, Brachioradialis; D, Recurrent vascular loop; E, Musculocutaneous nerve; F, Radial artery; G, Supinator; H, Posterior interosseous and radial nerves.

finger distally along the outer side of the tendon till it meets the resistance of the recurrent vascular loop (*Fig.* 324). Hook this up gently on the finger: divide and tie it. Mobilize the three long muscles which flank the outer

face of the forearm. Detach the flat tendon of the brachioradialis from its insertion into the base of the radial styloid. Flex the elbow through 90°, and retract the muscles widely out to expose the supinator.

Return to the biceps tendon: keep the knife close to its outer face and cut down upon the radial tuberosity (*Fig.* 325). This cut divides the

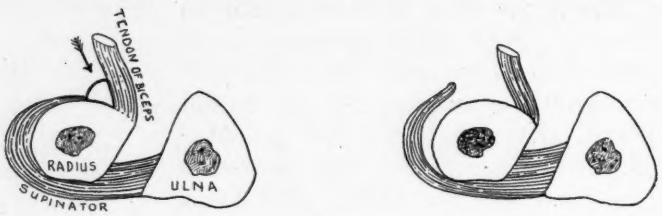

FIG. 325.—Showing the cut made through the bicipital bursa. Keep the knife close to the outer face of the biceps tendon. The supinator is then peeled off the radius. The arrow shows the direction of the cut through the bursa.

bicipital bursa, and the knife strikes the radial tuberosity where it lies in a bay formed by the supinator edge (*Fig.* 322). From this strategic point the rugine peels the supinator muscle off the bone. The muscle is turned out-

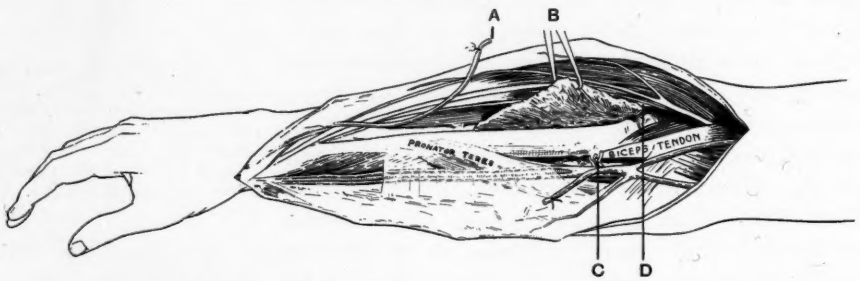

FIG. 326.—Completion of the exposure by putting the forearm in full pronation. A, Radial vein; B, Forceps retracting reflected supinator; C, Recurrent vascular loop (cut); D, Posterior interosseous nerve.

wards, carrying within its substance the posterior interosseous nerve. Put the forearm into a position of full pronation, and the radius will be revealed from end to end (*Fig.* 326).

My best thanks are due to Professor E. J. Evatt, D.S.O., for the opportunity of working out the details of this exposure in the School of Anatomy, Royal College of Surgeons, Ireland, and to Mr. Matthew Barry for his drawings.

DESQUAMATIVE AND DYSGENETIC* EPITHELIAL HYPERPLASIAS IN THE BREAST:

THEIR SITUATION AND CHARACTERISTICS: THEIR LIKENESS TO LESIONS INDUCED BY TAR.

By Sir GEORGE LENTHAL CHEATLE, London.

Some critics have declared that they can attach no importance to my observations upon desquamative epithelial hyperplasia in breasts, for two reasons: first, that it so frequently occurs in the breasts of women who have died of maladies unconnected with these glands; and, second, that it is also commonly seen in breasts containing other swellings which have been removed from living women. The second reason seems to me to contradict the importance my critics give to the first, for the second reason suggests what I believe to be true, viz., that there is a definite though distant connection between desquamative epithelial hyperplasia and the genesis of swellings that occur in the breasts of living women.

These facts are common knowledge, but I do not agree that because desquamative epithelial hyperplasia of the breast is so commonly seen in these circumstances, its presence is therefore unimportant. On the contrary, I believe it to be a pathological state within the actual lesions of which many tumour formations begin. The situation which desquamative epithelial hyperplasia occupies, the pathological changes that occur in it, the great length of time it persists, and the genesis of these additional changes therein, make its presence one of menace and importance. The site of its incidence is important, because it is situated mainly in the acini and terminal ducts, where also occur many other forms of epithelial hyperplasia, including a type of carcinoma of a very definite character, all of which I shall describe in this paper.

The importance of the time factor is this: Carcinoma beginning in a part where irritation is present, or even where irritation has been applied, may take a long time to mature. The period necessary for the induction of tar carcinoma in human beings is from twenty to thirty years. In connection with this fact, there are two more facts to which I must draw attention: (1) Desquamation of epithelium, papillomata, a type of dysgenetic epithelial

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* I substitute the term 'dysgenetic epithelial hyperplasia' for that of 'non-desquamative epithelial hyperplasia', a term I have used hitherto. By 'dysgenetic epithelial hyperplasia' I mean one that results in the formation of epithelial cells that are growing pathologically, not one that is throwing off cells that probably are of no further importance, i.e., 'desquamative epithelial hyperplasia'. The term includes: (1) A state wherein the growing epithelial cells show nuclear hyperchromatosis, variation in size, and mitoses (*see States A and B, Class II*); and (2) One in which these cell changes are not present (papillomata).

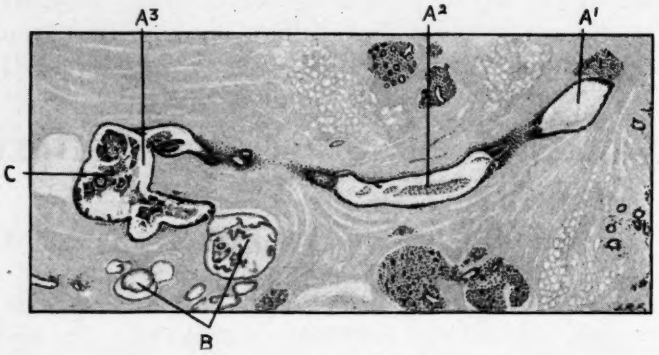


FIG. 327.—Part of whole section removed from female, age 34, for localized pain and nodularity of the lower and outer portion of the breast A^1, A^2, A^3 is the termination of a duct, B the acini into which it led. At A^2 desquamative epithelial hyperplasia is present, resulting in a collection of colostrum-like cells. At C in A^3 there is a multiradicular papilloma. At B desquamative epithelial hyperplasia of the acini has created a small cystic state.

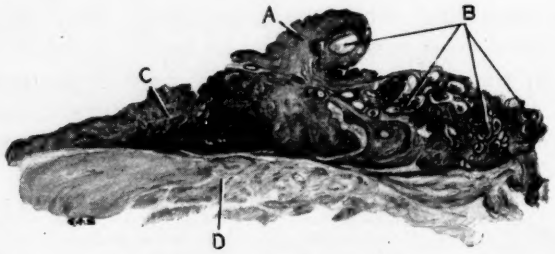


FIG. 328.—Whole section of breast removed from patient, age 41. A, Nipple. D, Pectoralis major. C, Normal portion of the breast. B, The only dilated duct in the breast. Its terminal branches are filled with the papillomatous tumours seen in *Fig.* 329. These terminal branches lead into acini dilated by sessile non-papillomatous dysgenetic epithelial hyperplasia. There is no epithelial-cell invasion in this breast. It is a case of my own, and my clinical diagnosis was carcinoma. There were no axillary lymphatic glands affected.

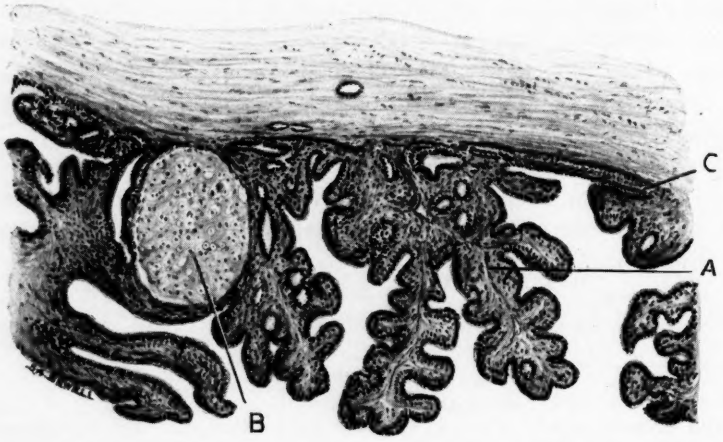


FIG. 329.—Taken from a duct of *Fig.* 328. C, Normal epithelial covering of the duct wall from which grows the papillomatous state seen at A, and which resembles the state of warts that occurs on the skin after the application of tar. B, Collection of colostrum-like cells that can be seen growing from the epithelial wall of the duct. In other parts of this duct the epithelial growth is sessile. The cells there show nuclear hyperchromatosis, mitosis, and they are irregular in size.

hyperplasia that does not invade outside tissues although histologically malignant, and carcinoma, all follow one after the other in this sequence before tar carcinoma appears in human beings (*see Figs.* 342, 343, and 344); (2) In the breast this sequence of events is precisely the same as that which I have just given in connection with tar cancer—viz., desquamative epithelial hyperplasia, followed by papilloma formation, which is in turn followed by a form of epithelial hyperplasia that is histologically malignant, but which in the absence of epithelial invasion cannot be said to be malignant (*State A, Class II*), and finally carcinoma (*see Figs.* 327–340, 345, 347–349, 351–353). These events can be seen occurring frequently in the lesion of desquamative epithelial hyperplasia. In producing these facts I have done enough to place desquamative epithelial hyperplasia in the position of initial importance.

It would not be correct to say that I propose excision as the only remedy. My observations entitle me to say only that many years may pass before more important and various pathological changes are induced in the lesions of desquamative epithelial hyperplasia, and that in any of these pathological changes, when once induced, intercurrent impulses may arise that arrest or prolong them, or hasten them into carcinoma.

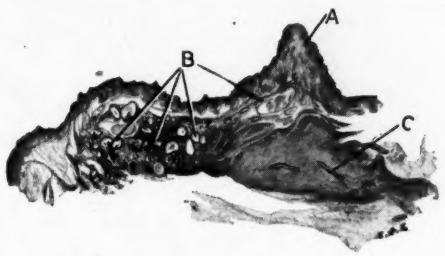


FIG. 330.—Whole section of a breast removed from female, age 36, for irregular nodularity in the lower and outer part of breast. Compare the whole section of *Fig.* 328, which belongs to the same type of pathological change. A, Nipple. C, Normal portion of the breast. B, Part of the only dilated duct in the breast. The terminal branches of it contained papillomatous multiple multiradicular tumours (*see Figs.* 331, 332, and 333). The terminal ducts led into many acini that were distended by non-papillomatous dysgenetic epithelial hyperplasia.

However, I have described other changes that are not to be discovered frequently in the breasts of women who have died of other diseases; in fact their detection in post-mortem breasts is decidedly uncommon. As it is to these changes I wish to draw more particular attention, an epitome of my observations that bear on all relevant pathological changes (including desquamative epithelial hyperplasia) will enable me to make my demonstration clear to my readers. In this paper I divide these pathological conditions into two classes:—

Class I.—Desquamative epithelial hyperplasia, i.e., an epithelial hyperplasia that results in a collection, within acini and terminal ducts, of cast-off epithelial cells which are probably of no danger to the individual. *Class I* contains those forms of epithelial hyperplasia to which I alluded in the first paragraph of this paper.

Class II.—Dysgenetic epithelial hyperplasia, i.e., one that results in a collection of living and pathological epithelial cells which may lead to three separate conditions:—

1. A condition in which the epithelial cells retain their normal structure; it produces papillomata confined within the acini and terminal ducts.

2. An epithelial hyperplasia in which the individual cells are showing signs of beginning malignancy—e.g., nuclear hyperchromatosis, variation in the size of cells, and mitosis—yet the process is still confined within acini

and terminal ducts and has not extended into the surrounding connective tissue (*State A, Class II*).

3. A more advanced condition in which, in addition to the cell changes, the process has extended beyond the walls of the acini and terminal ducts and has invaded the surrounding connective tissue (*State B, Class II*).

The presence of one or more varieties of dysgenetic epithelial hyperplasia in the same specimens in which early or late carcinoma is present is most significant (*see Figs.* 346–355).

All the specimens I reproduce as examples of these two classes were taken from living women. To *Class I* belong those changes that are so frequently discovered in breasts of women who die of maladies unconnected with breast

FIG. 331.—Taken from terminal duct and acini of *Fig.* 330. In the last portion of a terminal duct at A there is a multiradicular papillomatous tumour. This leads into the acini B, which are in an early cystic state.

disease. Although the specimens that belong to *Class II* have been removed from living women, these women might have died from maladies unconnected with breast disease while the lesions I reproduce here were present. Hence the occasional post-mortem discovery of these lesions in such circumstances ought not to create surprise, nor should it lessen their importance from pathological and practical standpoints.

Class I.—DESQUAMATIVE EPITHELIAL HYPERPLASIA.

Of this there are two types, *A* and *B*.

Type A.—Less common than *Type B*. In it there is a more or less completely generalized collection of desiccated, irregularly-shaped, ill-staining cast-off epithelial cells. The process is situated in the acini and terminal

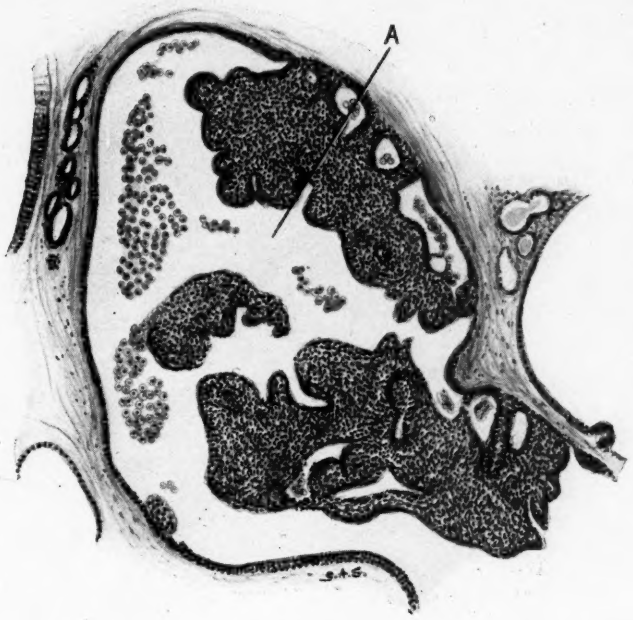


FIG. 332.—Another terminal duct from *Fig.* 330, and contains the same type of tumour. Growing from the wall of the duct can be seen a desquamative type of epithelial hyperplasia (A) resulting in the collection of colostrum-like corpuscles.

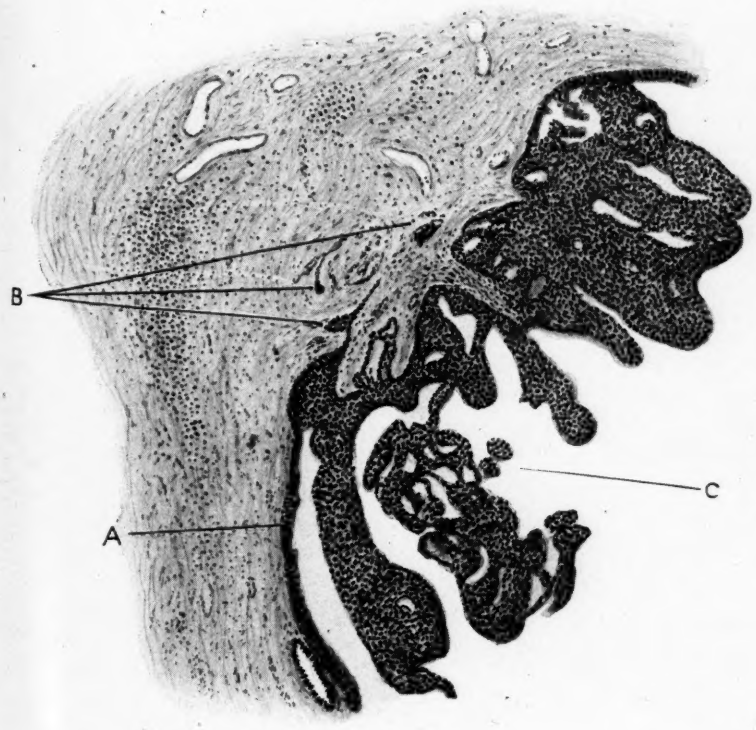


FIG. 333.—Drawing made from another terminal duct of *Fig.* 330. C, The papillomatous tumour it contained; A, Normal epithelium lining the duct; B, Histological appearances that suggest epithelial-cell invasion. Among these cells are some showing nuclear hyperchromatosis, mitosis, and irregularity in shape.

ducts. This type may remain many years without alteration in histological appearances. It is seen in a great many fibro-adenomata.

Type B.—Much more frequent and important than *Type A*. The process affects terminal ducts alone, acini alone, or it may appear as a combined affection of acini and terminal ducts (*see Fig.* 327). All varieties of *Type B* may be present in the same breast. In so far as it affects terminal ducts and acini, it resembles in situation *Type A* and the types of dysgenetic epithelial hyperplasia described in *Class II*. *Type B* is often a much more localized affection, and I feel sure it is much more important than *Type A*, because many forms of epithelial hyperplasia complicate the actual lesion it creates. For example, see *Fig.* 327, where a terminal duct affected by *Type B* is represented. In it, there is a multiradicular papilloma that was clinically undetectable. (*See also Figs.* 328–340.)

FIG. 334.—Papillomatous tumour in a duct which leads directly into the terminal ducts and acini seen in *Fig.* 335. *Figs.* 334 and 335 illustrate the only lesions to be discovered in the breast, after cutting whole sections. There was Paget's disease of the nipple.

Type B is easily recognized. In the early stages in the terminal ducts and acini, the epithelium undergoes hyperplasia and becomes elongated and feathery in appearance. The terminal ducts can often be traced to only one main duct, which may be the only duct affected in the whole breast (*see Figs.* 328 and 330). Only in the ducts the desquamated epithelial cells appear like colostrum corpuscles, which can be seen in all stages of formation arising from among the feathery cells. When the affected ducts are larger than those that are terminal, the feathery character of the epithelial hyperplasia is absent, but the colostrum-like cells can be seen in all stages of formation arising from the ordinary cells that line the large ducts. On rare occasions the desquamated cells are seen to have separated parts of the normal walls of ducts, through which they are being pushed into the tissues immediately outside the ducts (*see Fig.* 341). From these particular cells probably no other evil need be anticipated. It is in the

lesions of *Type B* that the genesis occurs of the dysgenetic epithelial hyperplasias I shall presently describe.

Type B is specially a cause of duct and acinous cysts. I have pathological and clinical evidence to adduce which shows conclusively that suspected and unsuspected papillomata and carcinomata respectively, or both combined, occur in these cysts.

Both *Types A* and *B* are frequently accompanied by hyperplasia of the connective tissue that immediately surrounds the ducts and acini. Whether

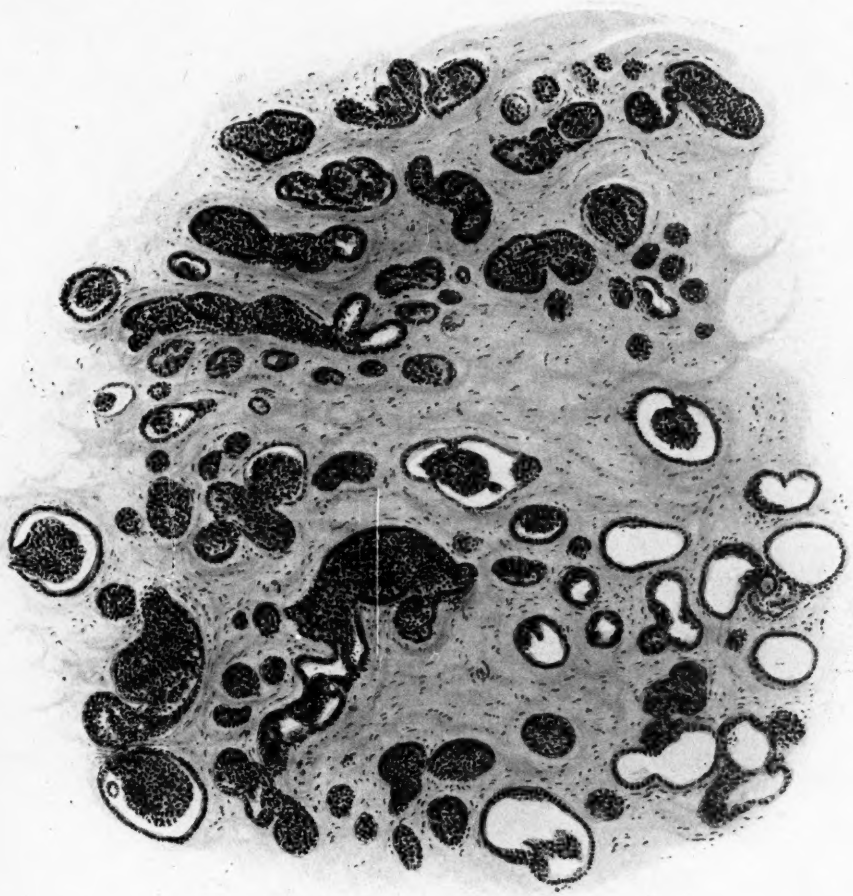


FIG. 335.—Termination of terminal duct of *Fig.* 334. In the ducts can be seen a mixture of papillomatous and sessile dysgenetic epithelial hyperplasia. Most of the acini are filled by an actively staining epithelial hyperplasia. There is no transgression of natural boundaries. Among the epithelial cells are mitotic figures, nuclear hyperchromatosis, and variation in shape.

this hyperplasia of the connective tissue is due to the same inducing agent as that of the epithelial hyperplasia, or whether it is only a response to the mechanical stretching induced by the dilating glandular elements, it is impossible to say. The pathological changes in *Class I* (specially *Type B* of

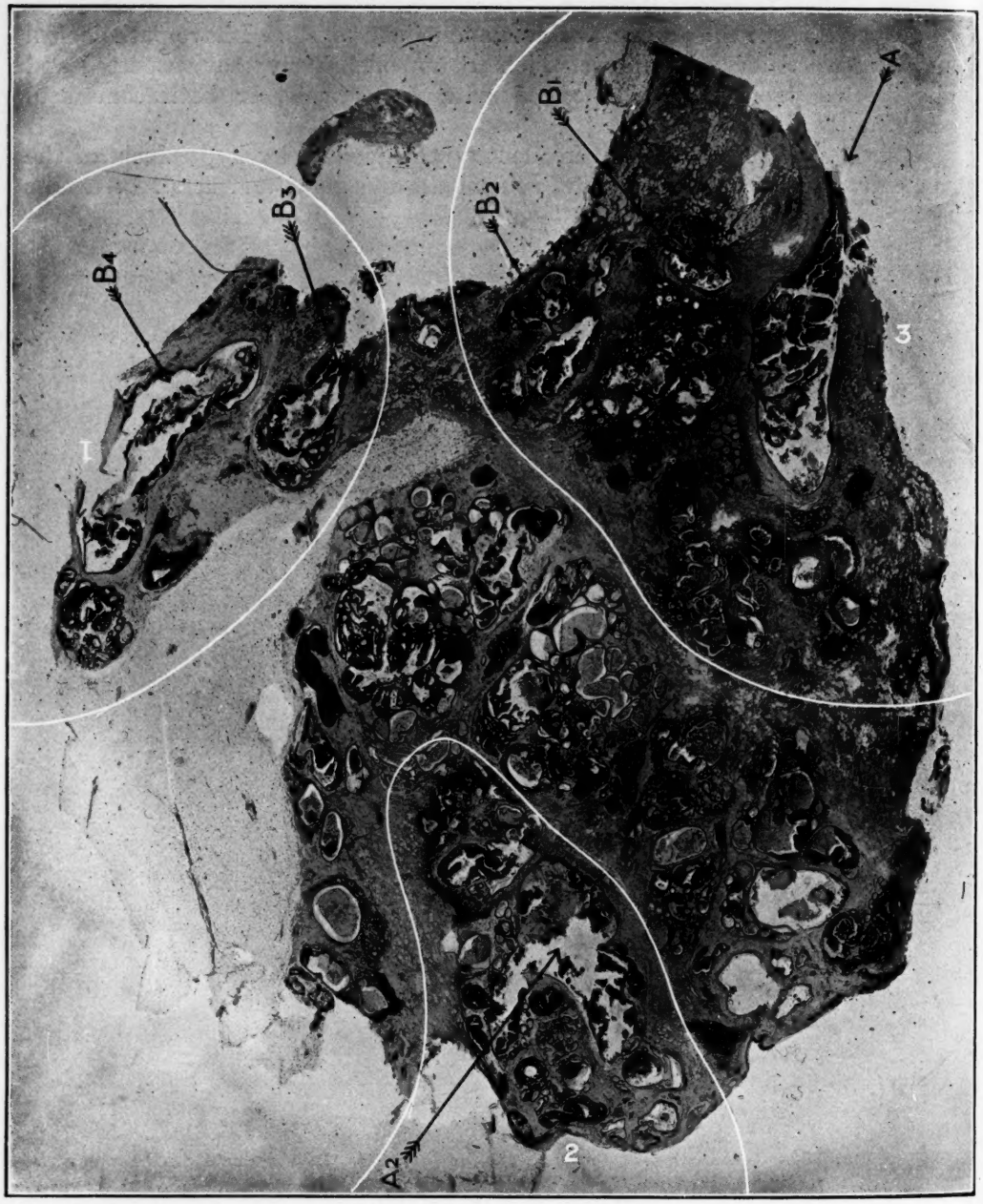


FIG. 336.—Section removed from lower part of breast of female, age 41. The provisional clinical diagnosis of a fibro-adenoma was made. Within the marked areas, at 1, 2, and 3, separate collections of terminal ducts and acini are filled by dysgenetic epithelial hyperplasia, which, in the ducts, is papillomatous. The only transgression of natural boundaries that occurred in this specimen was that the acinous cysts that were filled by epithelial cells were becoming confluent. Whole sections made of this breast revealed the fact that these terminal ducts led into one main duct, which was the only part of the breast affected. A, A_2, B_1, B_2, B_3, B_4, *see text*, p. 529.

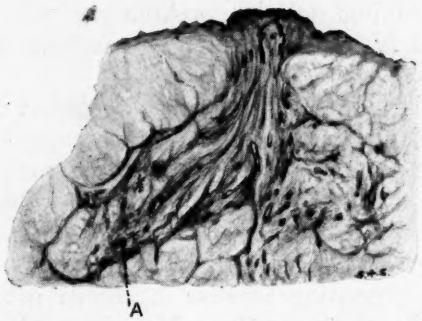


FIG. 337.—Breast of a woman, age 52. There was Paget's disease of three months' duration on the top of the nipple. A, Part from which *Fig.* 338 was taken.

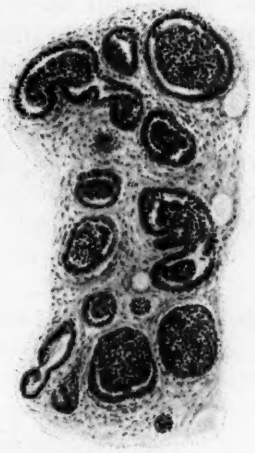


FIG. 338.—The only part of the breast in *Fig.* 337 that showed any dysgenetic epithelial hyperplasia. The terminal ducts are filled with papillomatous epithelial hyperplasia, and the acini are filled by growth that is not papillomatous. There was no transgression of normal boundaries.

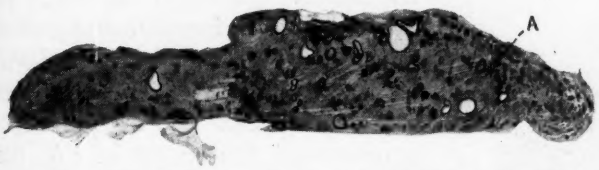


FIG. 339.—Whole section of a breast, from which Mr. G. L. Keynes allowed me to cut this section. I did not examine the whole breast. At A is the only part in the portion of the breast I received in which I could find dysgenetic epithelial hyperplasia, seen in *Fig.* 340.

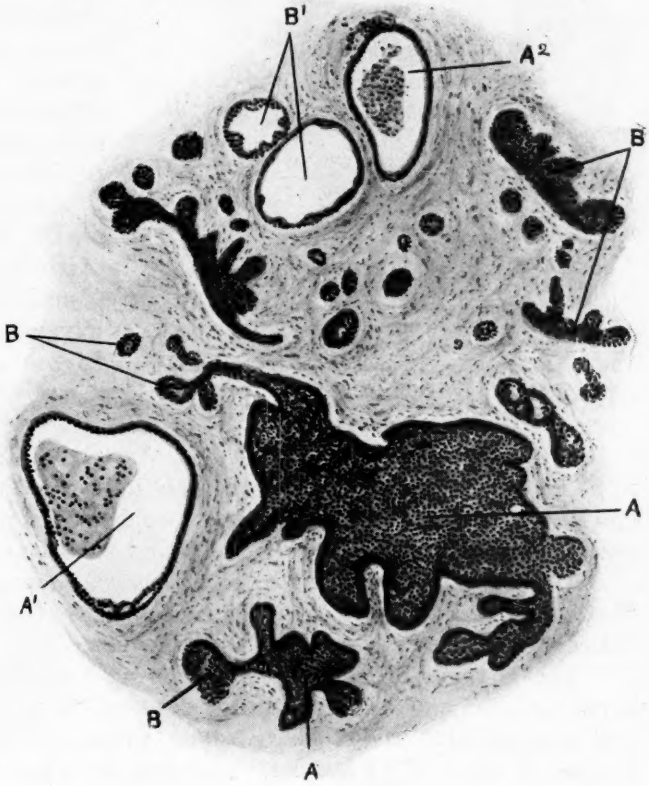


FIG. 340.—From A in *Fig.* 339. One terminal duct and its branches (A) were affected by sessile dysgenetic epithelial hyperplasia, in which nuclear hyperchromatosis, mitoses and irregularity of shape are marked. B represents the same type of hyperplasia in the acini. There was no invasion by epithelial cells in this specimen. The duct A^2, which is filled by colostrum-like cells as the result of epithelial hyperplasia, leads into the acini, B^1, which are in the early state of cystic formation. A^1 is another portion of a terminal duct which is undergoing the same process as seen in A^2.

that class) can be identified as playing definite preliminary parts in the sequence of events included in *Class II*, and it is thereby rendered a class of pathological and practical importance.

This sequence of events, beginning in epithelial hyperplasia that desquamates, passing on to epithelial hyperplasia that does not desquamate, but which gives rise to living pathological epithelial hyperplasia that may end in carcinoma, is beautifully brought out by *Figs.* 342, 343, and 344, which have been kindly lent to me by Dr. J. A. Murray, and represent the results, at different stages, of the application of tar to the skin of animals.

Take *Fig.* 342. Here the desquamating process is shown after a few applications of tar to the skin; horny layers of the epidermis are being piled up on one another as the result of desquamative hyperplasia of the superficial epithelial layers; the deepest layer is inactive and strictly limited to its normal boundaries. The behaviour of this skin corresponds to that of desquamating processes of the breast in *Fig.* 327, A, and *Fig.* 341.

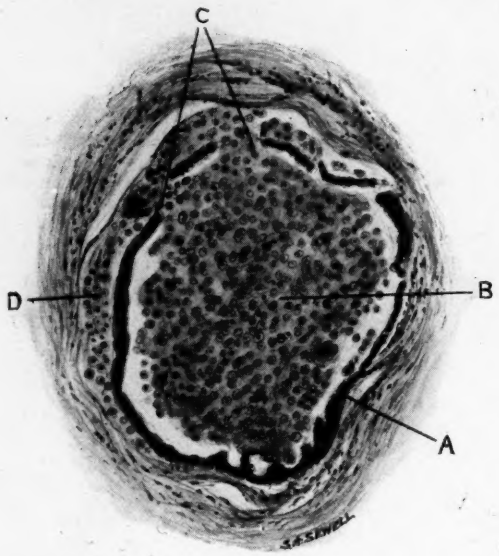


FIG. 341.—Transverse section of a duct the epithelial cells lining which are undergoing desquamative epithelial hyperplasia. A, Wall of the duct. B, Colostrum-like corpuscles filling and distending the duct. At C the epithelial wall has given way, and these colostrum-like corpuscles can be seen emerging from the duct and occupying the surrounding tissue outside it (D).

The second lesion in sequence is the formation of warts, and is dysgenetic in type. Epithelial cells removed from these warts and placed under the skin of the same animal either atrophy or form implantation cysts. These warts correspond to the formation of papillomata in the breast.

Fig. 343 represents the third of Dr. Murray's experiments. The epithelial hyperplasia is also dysgenetic in type, and is a result of a longer application of tar to the skin. Living epithelial cells are being accumulated which are capable of dangerous activity, as proved by the fact that Dr. Murray placed some of them under the skin of the animal, where they grew and metastasized. Yet the deepest layer of epidermis remains sacred to its normal boundaries. This condition corresponds to *State A*, *Class II*, in the breast.

The fourth experiment of Dr. Murray, shown in *Fig.* 344, is so obvious that no further allusion to it is necessary, except to demonstrate the final catastrophe of carcinoma.

The changes observed in Dr. Murray's experiments and also those that occur on human skin constantly exposed to tar are identical in sequence and comparative duration to those processes in the breast included in *Classes I* and *II* (*Figs.* 327 and 341 of *Class I*, also *Figs.* 328–355 of

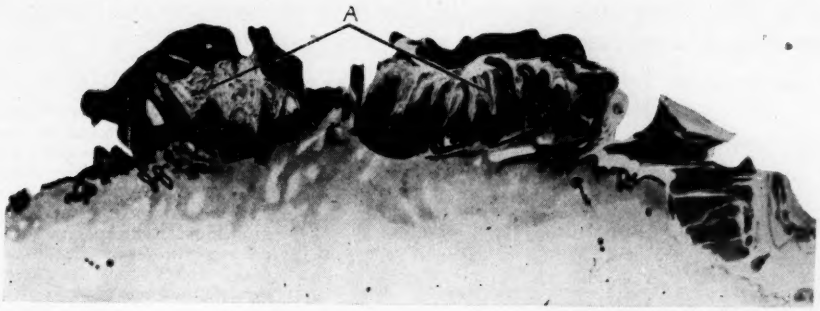


FIG. 342.—Skin of animal after few applications of tar. There is only a desquamative epithelial hyperplasia at A. (Dr. J. A. Murray kindly gave me leave to publish this and the two following pictures.)

FIG. 343.—Skin of another animal after more applications of tar than those administered to specimen in *Fig.* 342. The epithelial hyperplasia is dysgenetic, and cells taken from A were placed under the animal's skin, where they grew and metastasized. B shows that the normal boundaries of the epidermis have not been transgressed. (Dr. J. A. Murray's experiment.)

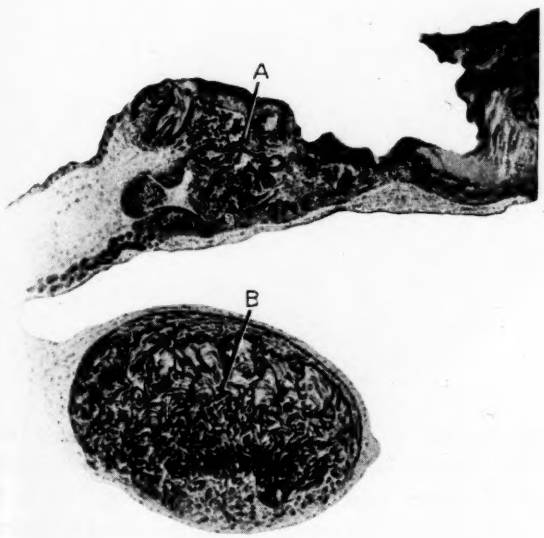


FIG. 344.—Skin of another animal after still more applications of tar. Carcinoma exists at A. B is a lymphatic gland enlarged and filled with epithelial cells from A. (Dr. J. A. Murray's experiment.)

Class II). The desquamated cells in the first of his experiments are thrown off from the skin surface into the air. Desquamated epithelial cells of the

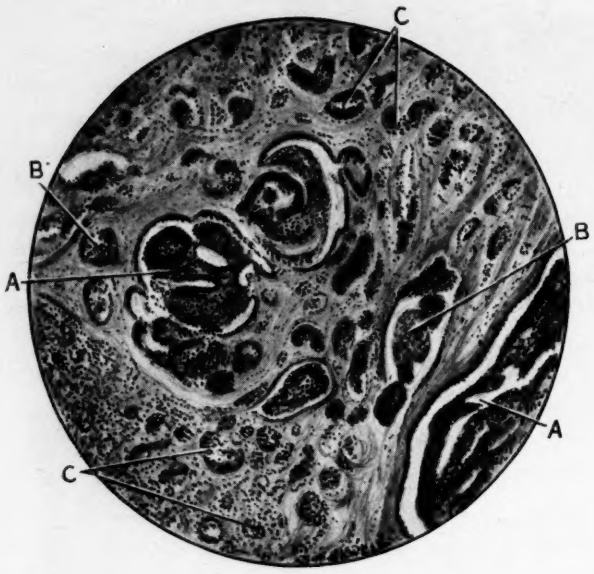
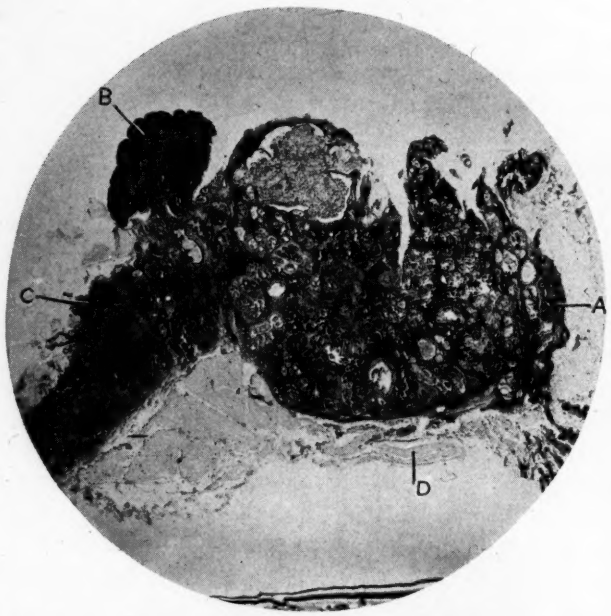


FIG. 345.—Section of a terminal duct (A), and some of its acini (B). At C epithelial cells have invaded structures outside the ducts and acini. The dysgenetic epithelial hyperplasia in the duct A is papillomatous. The specimen is taken from a breast of a female, age 42, who, eight years before its removal, had complained of pain in the affected breast. Four years before its removal, there was a discharge of blood from the nipple, the only occasion on which blood had been noticed. A few months before its removal, the breast became more painful. There were small multiradicular papillomata beginning in the ampulla and occupying the whole duct and its terminal branches. It was the only duct affected. The growth in the acini is not papillomatous. There were no lymphatic glands enlarged in the axilla. (Dr. W. R. Smith's patient.)

breast must collect in and distend the affected ducts and acini (*see Figs.* 327 and 341). The third of Dr. Murray's experiments (*see Fig.* 343) is the

FIG. 346.—Whole section of breast removed from woman, age 47. Half of the section is occupied by a carcinoma (A). The other half (C) is normal except for a few small cysts just outside the periphery of the photograph. B, Nipple. D, Pectoralis major. There were large discrete lymphatic glands in the axilla containing epithelial cells. The woman died two years after removal of the breast and axillary lymphatic glands. The main size of the tumour was due to the enormous dysgenetic epithelial hyperplasia that was contained within dilated ducts and acini (*see Figs.* 347 and 348). There was marked epithelial cell invasion among the tissue surrounding the affected ducts and acini.



most deeply interesting of the four. As I have already pointed out, he took away some of the cells from their position at A, and placed them under the skin of the animal, where they grew and metastasized. Is it to be inferred that those dysgenetic epithelial cells, in their undisturbed position

at A, were malignant, or did they become malignant only when they were removed and inserted into tissues outside the epidermis? The importance of this point lies in the fact that a dysgenetic epithelial hyperplasia in the

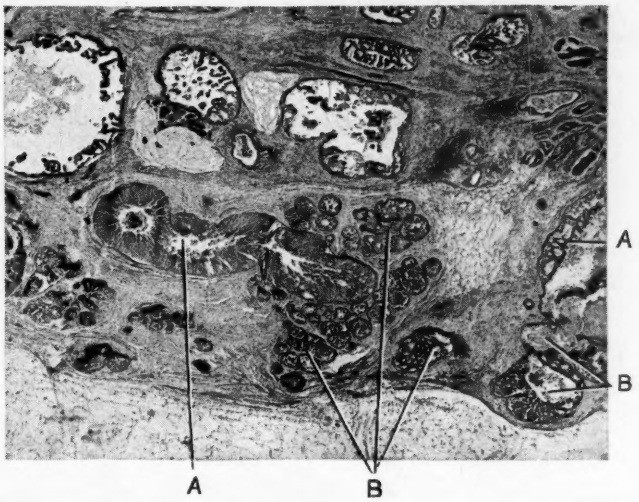


FIG. 347.—Photograph taken by Mr. Walter Barnard of the edge of the tumour in *Fig.* 346. The ducts A, A were surrounded by normal elastica, and led into the acini B, B. The dysgenetic epithelial hyperplasia in all these structures was non-papillomatous. The epithelial hyperplasia is contained within normal boundaries.

breast gives rise to a most suspicious-looking collection of living cells, while they are strictly confined within ducts and acini (*see State A, Class II*).

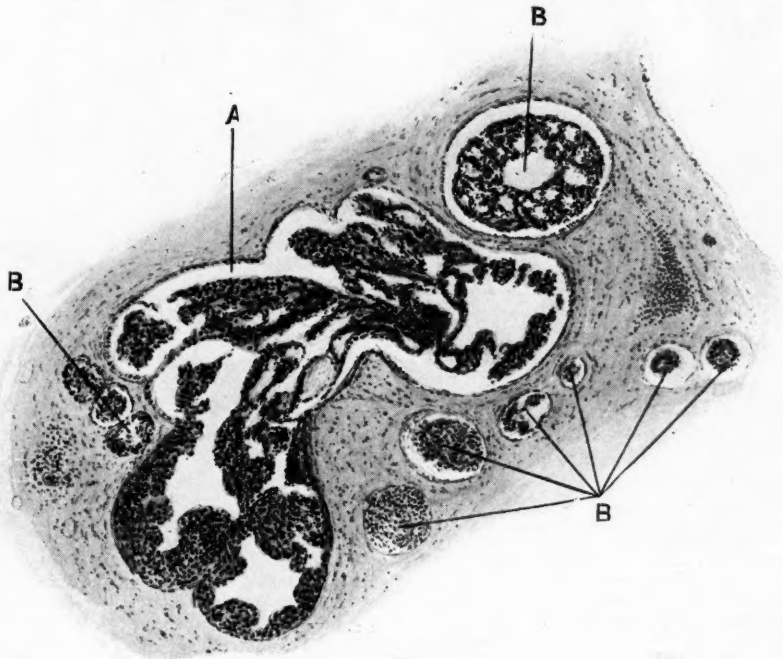


FIG. 348.—Drawing of acini and terminal ducts made from the centre of the tumour in *Fig.* 346. The elastica of the duct A was intact. The dysgenetic epithelial hyperplasia in the duct is papillomatous. The growth in the acini B, B, B is not papillomatous. Like the epithelial hyperplasia in *Fig.* 347, it is strictly confined to normal boundaries, although taken from the carcinomatous tumour in *Fig.* 346.

Here the following question arises. Should these dysgenetic epithelial cells of the breast gain admission into outside tissues by traumatic, chemical, or some other means, would they be capable of multiplication and metastasis? I believe they would behave in this way when they appear active and not degenerating, and, in spite of an absence of epithelial-cell invasion, I consider them to be potentially malignant, a belief that is supported by the fact that

FIG. 349.—Drawing made from the centre of a carcinomatous tumour in the breast of a woman, age 51. 1, 2, Terminal branches of the same duct, surrounded by elastica. 4, 5, Acini. 3, Area in which epithelial cells have invaded surrounding structures. The dysgenetic epithelial hyperplasia in the duct (1, 2) is papillomatous. The growth in the acini is not papillomatous. The lymphatic glands in the axilla contained carcinoma, and the patient died four and a half years after the operation. The epithelial cells in the duct show nuclear hyperchromatosis, mitosis, and irregularity in shape, corresponding thereby to the epithelial cells in *Figs.* 328, 330, 333, 334, 340.

precisely similar histological appearances are seen in the ducts and acini of tumours where epithelial cells have escaped from their normal boundaries and constitute a state of carcinoma (*see* the ducts and acini in carcinomatous tumours in *Figs.* 345–354).

I admit that at present it is impossible to determine absolutely whether a microscopical preparation demonstrates the presence of carcinoma, unless it shows a dysgenetic epithelial hyperplasia from which epithelial cells have

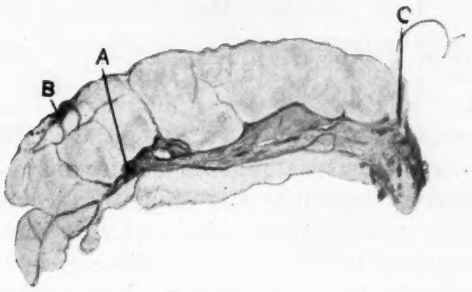


FIG. 350.—Drawing from a whole section of half a breast that was removed twenty years ago for the purposes of diagnosis. The woman was 50 years old, and is still alive. The tumour (A) is the smallest carcinoma I have removed from a breast. C, Position of the nipple. B, A small portion of skin covering the gland. There were no axillary glands affected.

invaded surrounding structures, where they multiply and undergo metastasis. Yet in the earliest stages of the carcinomatous process there must be one in

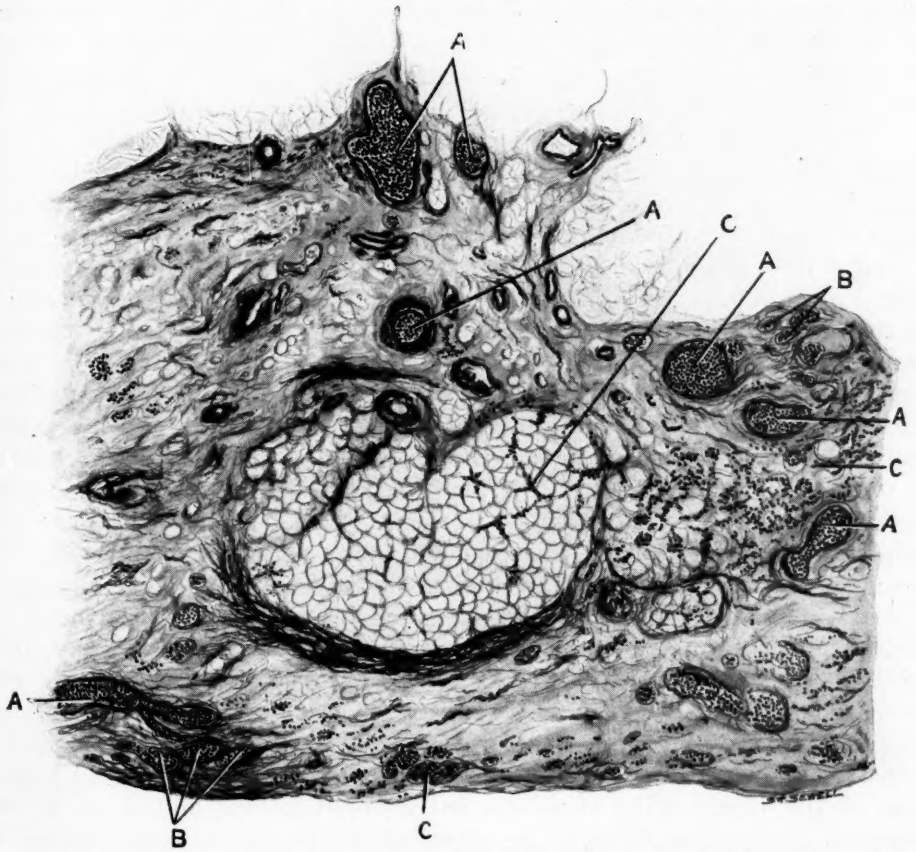


FIG. 351.—Drawing made from the tumour A of *Fig.* 350. A, Terminal ducts surrounded by their elastica coats. B, Acini. C, Epithelial-cell invasion of outside structures, including adipose tissue. The dysgenetic epithelial hyperplasia in the ducts and acini is not papillomatous.

which a dysgenetic epithelial hyperplasia is strictly confined within ducts and acini. Such a change in the sequence of events is demonstrated by definite signs of malignancy as indicated by the character of the individual tumour

cells, which show nuclear hyperchromatosis, variation in size, and mitoses, although the process is still confined within ducts and acini (*State A, Class II*).

It is inevitable that difficulties should arise, when conjectures upon histological appearances have to be made without the aid of that biological evidence that could be attained only by experiment conducted upon human beings. The solution of these difficulties by this means is therefore impossible. Is it justifiable to fall back upon the evidence afforded by experiments conducted on animals? Whatever be the answer to this question, it is of extraordinary interest to study the animal experiments and compare their various stages with the changes that occur in the breasts of human beings. The time required to induce tar carcinoma in mice occupies a quarter or more of the animal's lifetime, during which period the skin to which the tar has been applied has gone through a sequence of pathological changes to which I have already drawn attention. An important point to realize is that in any one of these changes the process may be arrested, and the genesis of carcinoma may not mature. It is a fact that precisely the same pathological changes take place in the breast, and in the same sequence, viz., the desquamative hyperplasia, especially *Type B* in *Class I*, the dysgenetic hyperplasia of *Class II*, in which occur papillomata, and the conditions indicated in the *States A* and *B* of *Class II*. Lastly, when carcinoma eventually matures in human breasts, the total period of its induction usually corresponds to a quarter or more of a lifetime of the individual.

Can it be said that the states in the breasts of human beings correspond, in histological appearance and periods of their development, with those states seen in mice during the induction of tar cancer? Similarity of histological appearances cannot be denied; but there are those who deny, with some reason, that a quarter of a mouse's lifetime corresponds to a quarter of the lifetime in human beings. Fortunately, it is unnecessary to argue this point, because Harold Barnard, years ago, showed that the exposure of the human skin to tar occupied from twenty to thirty years before tar carcinoma ensued, and this evidence is available among workers in tar at the present time. He also pointed out that during this period a state of things appeared in the following sequence: warts; a state that microscopically looked like squamous epithelioma, but which was not accompanied by epithelial cell invasion or metastasis, and from which recovery could take place; and, finally, true squamous epithelioma with invasion and metastasis. Hence, because the knowledge concerning the lesions induced by the application of tar to mankind is well established, it is unnecessary to fall back on experiments on mice for the important evidence that connects the changing types of epithelial hyperplasia of the breast with those to be observed in the induction of tar carcinoma. I consider them to be identical in histological appearances, in the time occupied in their induction, in the similarity of their arrest in some instances, and in their slow or rapid progress in others. Hence I consider it wrong to regard the desquamative epithelial hyperplasia of *Class I* as being of no importance. I regard it as being a proemial state wherein an irritant remains in action undisturbed for a long time. In point of time it is too far away to be regarded as 'pre-cancerous'. There is at least one state that

occurs later in life than desquamative epithelial hyperplasia and is nearer to the carcinoma process (*State A, Class II*), and can with more reason be regarded as 'pre-cancerous'.

Class II.—DYSGENETIC EPITHELIAL HYPERPLASIA IN ACINI AND THE TERMINAL DUCTS WITH WHICH THEY COMMUNICATE.

I do not intend to refer to purely duct carcinoma, which naturally belongs to this class. Neither shall I allude to duct papillomata, further than to point out that they occupy in sequence of breast events the same position

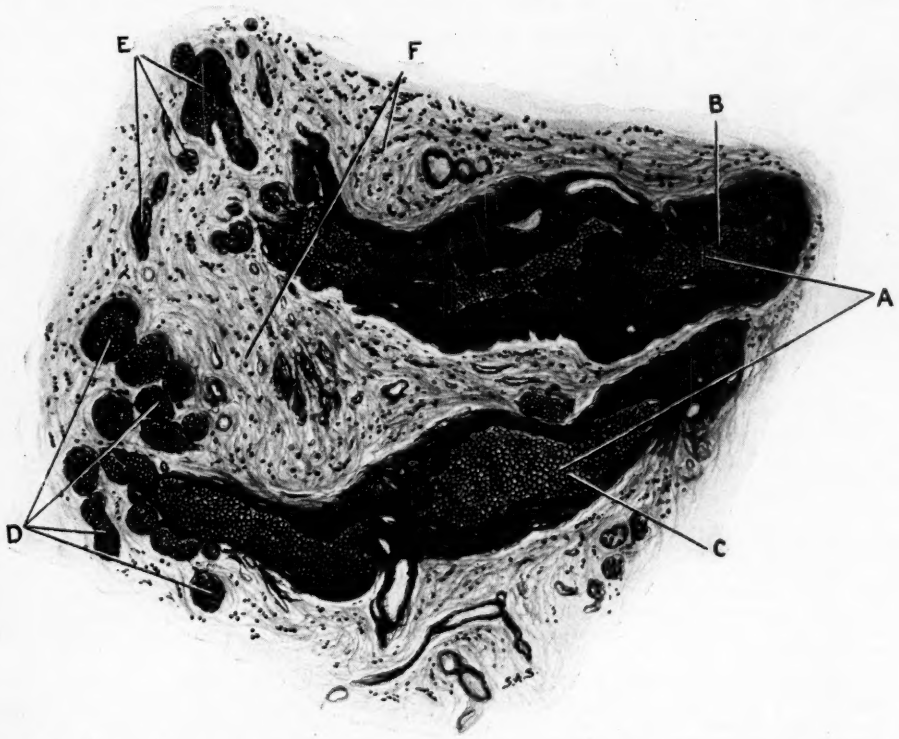


FIG. 352.—From the breast of a woman, age 38. Longitudinal section of terminal ducts, B, C, made just below bifurcation of a larger duct at A. D, E, Acini belonging to respective ducts, C and B. The elastica has been stained and has undergone enormous hyperplasia. There is marked epithelial cell invasion of tissues (F) outside the ducts and acini. The dysgenetic epithelial hyperplasia in duct and acini is not papillomatous. The tumour was not bigger than a finger-nail and occupied the outer part of the breast, between the nipple and the periphery of the gland. There was no affection of the lymphatic glands in the axilla. The Wassermann reaction was negative. (Mr. Percy Legg's patient.)

as that occupied by warts in the skin after the application of tar. I refer only to two kinds of dysgenetic epithelial hyperplasia (*States A* and *B*) that affect acini and the terminal ducts communicating with them. Before proceeding, it is essential that I should say that I do not consider that carcinoma of the breast of purely acinous origin has been demonstrated. In the earliest carcinoma in breasts (not purely of duct origin) I have never seen

a dysgenetic epithelial hyperplasia of an acinus that has not been accompanied by a similar process in the terminal ducts with which the affected acinus communicates. In many examples of early carcinoma the terminal duct implication of this kind is purely papillomatous in type, arising from the actual duct epithelium (*see Figs.* 345–349). This fact renders it impossible to infer that the duct is acting merely as a pathway by which the growth is spreading from the acinus. There is another fact that renders this inference impossible. It is this. I can show ducts (*Fig.* 354, for example) in which a

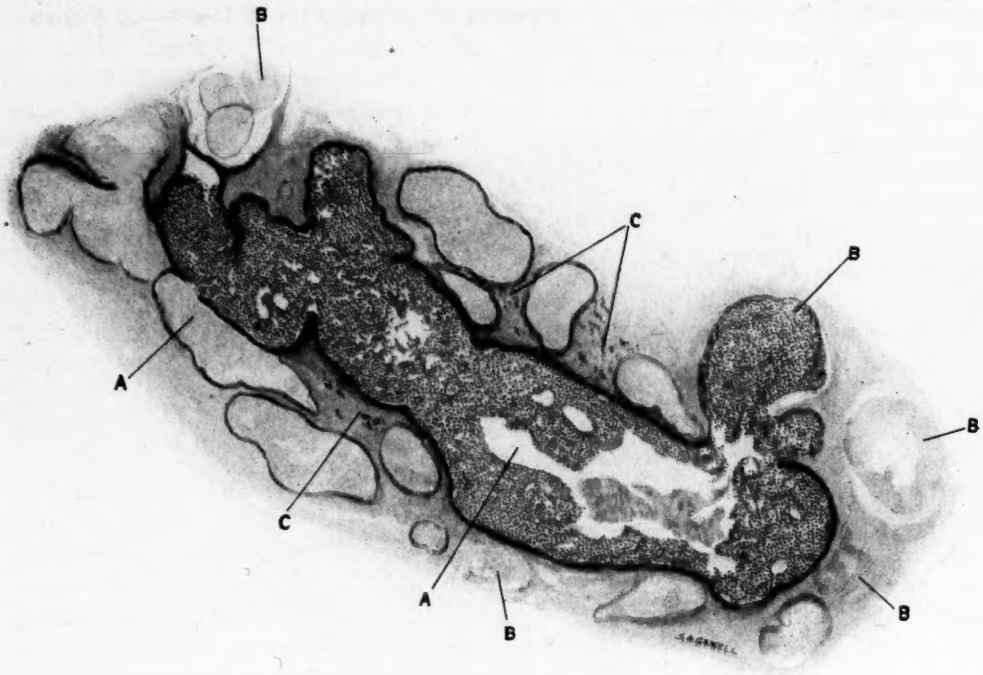


FIG. 353.—Longitudinal section of a terminal duct, A, A, and its acini, B, B, B, B. The elastica has been stained. The dysgenetic epithelial hyperplasia in only the main duct and one acinus has been drawn to show how full they are of growth (not papillomatous in type). The shaded ducts and acini were full of the same growth, which in all these structures was strictly confined within normal boundaries. This duct and its acini were discovered in the centre of an advanced carcinoma of the breast. At C, C there is epithelial-cell invasion of outside tissues. The woman was 49 years old, and died of the disease two and three-quarter years after the operation.

non-papillomatous dysgenetic epithelial hyperplasia is occurring that directly communicates with normal acini. The disease, in each instance, must be hyperplasia of duct epithelium as well as a hyperplasia of acinus epithelium, when the disease is present in the latter structure. One may have begun before the other, but as I have not seen a purely acinous affection in carcinoma of the breast, I cannot say in which structure the disease first began—although all my work indicates that it first appears in the duct.

Frequent examples of the histological appearances of *Class II*, which I am about to describe, are to be observed in breasts where there is early

Paget's disease of the nipple. This disease, when it affects the nipple, is commonly complicated by the development of primary carcinoma in the ducts or terminal ducts and acini in the breast. Hence one would expect to see either dysgenetic forms of epithelial hyperplasia immediately antecedent to carcinoma (*State A*), or early carcinoma itself (*State B*). It is essential in this investigation that all specimens prepared for microscopical examination should be stained to show the elastica. In the early stages of dysgenetic epithelial hyperplasia the elastica has not been destroyed—by distention or otherwise—and its presence is invaluable in enabling the observer to distinguish between ducts and acini and between ducts and lymphatic vessels and spaces.

I divide *Class II* into two states: *State A*, in which the dysgenetic epithelial cells are strictly confined within terminal ducts and acini; and

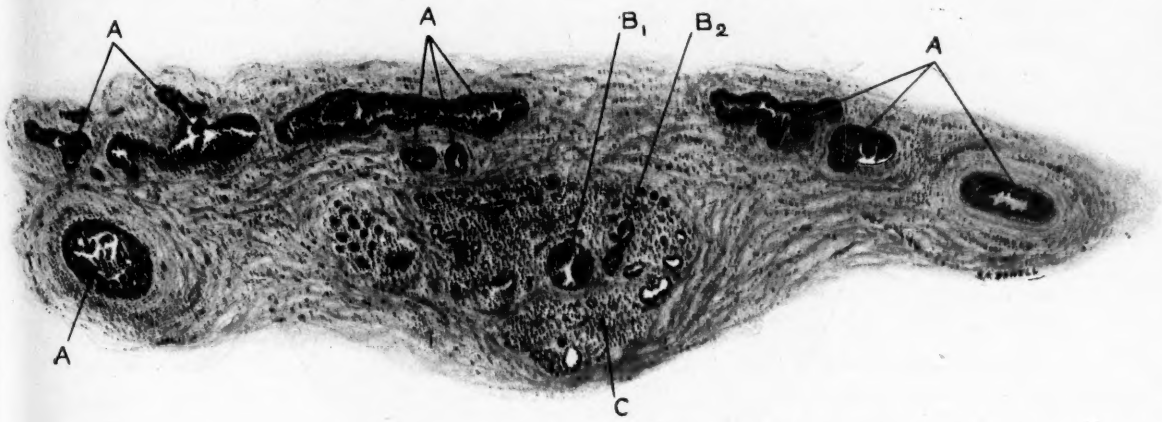


FIG. 354.—A, Longitudinal section of a convoluted duct. C, A lobule in which branches of the duct terminate at B_1 and B_2. The elastica is not stained in this section, but it was in other sections, and had undergone enormous hyperplasia throughout the whole breast. The figure is part of a whole section of the gland. The ducts show a dysgenetic epithelial hyperplasia, not papillomatous, and strictly confined within normal boundaries. There was no epithelial growth in any acini. The woman was 38 years old. There is epithelial cell invasion near this part of the gland, and no other focus of dysgenetic epithelial hyperplasia could be found in the breast than that which exists in this duct. The lymphatic glands in the axilla did not contain carcinoma. The Wassermann reaction was negative.

State B, in which the dysgenetic epithelial cells have at some part, or parts, escaped from the affected terminal ducts and acini, and have appeared in the surrounding connective tissue. Metastasis may have also occurred, and a state of carcinoma have been established.

State A.—Figs. 328–340 and 355 represent the histological appearances of *State A*, and are taken from seven different patients. In all of them only terminal ducts and acini are the parts affected by dysgenetic epithelial hyperplasia. *Figs.* 328 and 336 are the biggest lesions I have of this state. In other specimens, where the same tumour formations in the ducts and acini have been larger than this, they have always been carcinomatous (*see Figs.* 346 and 349) and belong to *State B*. The lesions in *State A* usually occur in younger women than the early carcinomatous lesions described in *State B*.

The significance of these two facts may be that *State A* is a state from which carcinomata (*State B*) may emerge.

The acini in *State A* are all of the same histological appearance. They are filled or partially filled by sessile dysgenetic epithelial growth, and only occasionally is there a suggestion of papillomatous formation. The ducts, however, do not always present the same histological appearances. In *Figs.* 328–336 the dysgenetic epithelial growths in the ducts are papillomatous, and in those of *Figs.* 340 and 355 the growths of epithelial cells contain no fibrous-tissue elements.

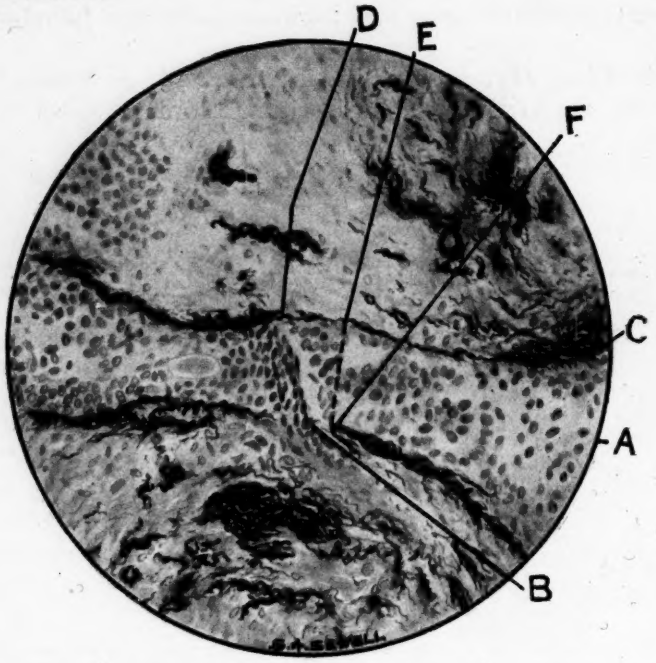


FIG. 355.—A, Longitudinal section of terminal duct. C, Elastica, stained. At B epithelial cells are emerging from the interior of the duct into the outside tissues. The dysgenetic epithelial hyperplasia in this duct is not papillomatous in type. There is enormous hyperplasia elastica in other parts of this breast, and the Wasserman reaction was positive. D, E, F, *see text*, p. 530. From a woman, age 42.

In the vast majority of the affected ducts and acini in these breasts in *Class II*, *State A*, there is not the slightest suspicion on microscopic examination, of epithelial cells that have transgressed their normal boundaries. Yet I regard *State A* as being second in importance only to carcinoma itself. Consequently it may well be asked, if I consider that to be so, why is not an intermediate stage between *States A* and *B* more demonstrable than in any specimen I have shown. In reply I would draw attention to *Fig.* 333, reproduced from *Fig.* 330, where there is a state of things that can be explained by an epithelial cell invasion; but as these histological appearances are open to other explanations, I do not reproduce them as an example of epithelial invasion. Further, the examples I have given in *State B* are such early stages of carcinoma that I consider they may be regarded as being almost within the realm of a transitional stage. *Figs.* 327, 329, and 332

illustrate the connection there is between *Type B* of *Class I* and *State A* of *Class II*. Colostrum-like cells are still present in these specimens. It might be said that these colostrum-like cells are a result of desquamation from the surface of the tumour, but there are no signs of their being formed from the cells at this part of the tumour, and they can be seen developing from otherwise normal cells of the duct wall. However, it is a fact that colostrum-like cells are sometimes thrown off by desquamation in all kinds of dysgenetic epithelial hyperplasia in ducts (*see Fig.* 328), but this fact may emphasize rather than disconnect the alliance that appears to exist between *Type B*, *Class I*, and *States A* and *B*, *Class II*. In describing *State A*, I wish particularly to analyse the appearances seen in *Fig.* 336. Very few seem able to realize, without its being pointed out, that A, A2, B1, B2, B3, and B4 are all terminal ducts containing papillomatous and sessile dysgenetic epithelial hyperplasia, and that they directly communicate with separate clusters of acini, which are distended by dysgenetic epithelial hyperplasia. In fact the terminal ducts and acini of one main duct alone are the only parts affected. The same appearances are seen in *Figs.* 328 and 330, which are cut from another angle. In many parts of *Fig.* 336 the acini, distended with growth, are becoming confluent. What could be the result of progress in the epithelial hyperplasia of *Fig.* 336 ? It might be said that the tumour may grow to an enormous size and be composed of cysts filled by epithelial cells and all contained within normal but grossly distended structures. An appearance such as this has never been seen, except in the presence of carcinoma (*see Figs.* 346 and 349). Hence I believe that the only result of progression in *State A* must be carcinoma ; a remarkable example of this process, as a progression from *State A* to *State B*, is that depicted in *Fig.* 346, where half the breast is occupied by a large carcinoma. The size of this tumour is mainly due to the dysgenetic epithelial hyperplasia that still occupies distended but otherwise normal boundaries (*see Figs.* 347 and 348, also compare with *Figs.* 349 and 353), and the increase of size of this tumour that can be allotted to infiltrating epithelial cells is remarkably small. The patient from whom this picture was photographed died of the disease two years after the operation. There can be no doubt that *Fig.* 336 and *Fig.* 346 form a remarkably connected pair.

Now, to call more particular attention to the sections of whole breasts reproduced in *Figs.* 328, 330, and 336. In these specimens, very interesting and diffuse dysgenetic epithelial hyperplasia can be seen. The growths are occurring in pre-existing cysts. Nobody can doubt that the terminal ducts and acini were dilated before the growth occurred. There are so many empty cysts in the neighbourhood of the growths, and the dilatation of the respective ducts at the nipples in *Figs.* 328 and 330 are so separated from the dilated terminal ducts and acini into which they directly lead, that it is impossible to assume that these dilatations near the nipple could have occurred as secondary processes. In the terminal ducts of the three specimens, the growths are a mixture of papillomatous and sessile dysgenetic epithelial hyperplasia. The remarkable fact which concerns these three specimens is that in each the terminal branches of only one main duct have been affected—a fact that has been demonstrated by cutting whole

sections, in series, of these breasts. The importance of the fact lies in the obvious inference that only one duct contained the irritation necessary to induce the hyperplasia. Although the growths are strictly confined, in most parts, within terminal ducts and acini, they give rise to very suspicious histological appearances.

In connection with the restriction which dysgenetic epithelial hyperplasia in *State A* shows to normal boundaries, I must refer to an important duct in *Fig.* 354. In it there is a dysgenetic epithelial hyperplasia, sessile (not papillomatous) in type. The peripheral half of this duct is filled and distended by living epithelial cells that have grown from its walls. I could not discover any acinous affection (*see* lobule C, *Fig.* 354). Serial sections of the whole of this breast did not reveal any other form of dysgenetic epithelial hyperplasia. Although I could not discover in any part of this duct a place from which epithelial cells were escaping, yet from this duct epithelial cells must have escaped to create the early state of carcinoma that was present in this breast. It is difficult to know whether to allot this duct to *State A* or *State B*. This difficulty of allotment emphasizes the importance of *State A*. Compare this specimen with the longitudinal section of the terminal duct from another breast, *Fig.* 355. At one point (B) epithelial cells are pouring out of the duct into the surrounding tissue, and the presence of carcinoma is certainly established. Now, suppose this duct had been cut and viewed transversely at the dotted line between E and F, i.e., in immediate juxtaposition to, but not including, the part where epithelial cell invasion is occurring, all that would have been visible in this imaginary transverse section would have been the distention of the duct by a dysgenetic epithelial hyperplasia; but, as there would have been no indication of cells passing through its walls, nobody would be justified in assuming it to be malignant; and yet it must be part of the carcinoma process, and a most intimate part, for exactly similar cells are in immediate contact with it invading the surrounding tissue. Further, what could be said of the epithelial cells at D? They must surely be malignant, or potentially malignant. I must leave it to the imagination and point of view adopted by my readers to make their own decisions on this matter.

I consider the dysgenetic epithelial hyperplasia, at the point of my imaginary transverse section, although the cells are strictly confined within the duct walls, to be malignant. I make the same conclusion concerning the epithelial cells contained in the affected ducts and acini in the carcinomata from which *Figs.* 345, 347–353 are taken. I believe that *State A* of *Class II* is a potentially malignant state, and that, should progress continue, the catastrophe of carcinoma is only a question of time. Whatever my belief is in this matter, there can be no doubt that *State A* is histologically more nearly connected with *State B* than any other pathological process in the breast; but no definite statement can be made as to whether it is only a fire prepared for the match which lights the conflagration of carcinoma, or whether it is a definite preliminary stage of carcinoma. Hence, if there must be the term 'pre-cancerous', I would apply it to this state. In making this statement, I would lay down as a law that there is no kind of epithelial hyperplasia that will inevitably end in carcinoma.

State B.—Here the dysgenetic epithelial hyperplasia is limited to acini and terminal ducts, and has precisely the same histological appearances as those in *State A*. Epithelial cells have invaded the surrounding tissues and may have undergone metastasis.

There is no doubt whatever that carcinoma exists in all the tumours I describe in *State B*. *Figs.* 345–355 are taken from early or advanced carcinomata removed from eight different breasts. In all of them the acini and terminal ducts are the only parts affected (except in the advanced carcinoma of *Fig.* 346, where two ducts as well as their acini are completely filled with dysgenetic epithelial hyperplasia from the ampulla downwards). The dysgenetic epithelial hyperplasia in the acini and terminal ducts are the same in histological appearances as those in *State A*. Sometimes the duct tumours are papillomatous, and sometimes purely sessile and non-papillomatous (*see Figs.* 351–355). As growth of the tumours proceeds, the intra-duct and intra-acinus tumours become more rampant (*see Figs.* 347, 348, 349, and 353, which were taken from three advanced carcinomata). Curiously enough, the tumours in the ducts and acini, although becoming rampant, still maintain their papillomatous or non-papillomatous state until all traces of duct and acinus walls are completely destroyed. The maintenance of papillomatous or non-papillomatous states respectively throughout the lives of these tumours indicates some method in their madness.

From some part of the dysgenetic epithelial hyperplasia epithelial cells have escaped, and they have invaded surrounding tissues and metastasis has occurred. It is difficult to detect the exact regions of ducts and acini from which the escapes of epithelial cells were made, even from an examination of whole sections cut in series. *Fig.* 355 was a lucky discovery.

SUMMARY AND CONCLUSION.

1. There appears to me to be a definite sequence of events in epithelial changes in the breast that end in carcinoma. In making this statement I do not wish it to be inferred that every event in the sequence invariably occurs—for instance, papilloma may be absent, and its place may be represented by a sessile and non-papillomatous state. One or more of these stages may be absent in the development of acute carcinoma.

2. It is most significant that the different epithelial changes that I have described have a common site of origin, namely, the acini and terminal ducts.

3. It is important to realize that the chain of events may be interrupted at any point, due either to intrinsic biological changes in the cells or to extrinsic factors such as variation in connection with the exciting cause. The interruption may result in the process remaining latent or in its subsequent degeneration and disappearance.

4. The sequence of events to which I have called attention begins as what I have termed a desquamative epithelial hyperplasia, and passes on to an epithelial hyperplasia which is not desquamative and which I have termed dysgenetic epithelial hyperplasia. Included in the latter, and following desquamative epithelial hyperplasia, are epithelial changes which occur in the following sequence :—

a. A condition in which the epithelial cells retain their normal structure and produce papillomata confined within the acini and terminal ducts.

b. An epithelial hyperplasia in which the individual cells are showing signs of beginning malignancy—e.g., nuclear hyperchromatosis, variation in size of the cells, and mitoses—yet the process is still confined within acini and terminal ducts and has not extended into the surrounding connective tissue (*State A, Class II*). This is the state which immediately precedes carcinoma, and hence is *the* 'precancerous state'.

c. A more advanced condition in which, in addition to the cell changes, the process has extended beyond the walls of the acini and terminal ducts and has invaded the surrounding connective tissue (*State B, Class II*).

5. The fact that one or more of the various epithelial changes, particularly the 'precancerous state', is found in the centre of fully developed carcinoma is most significant.

6. The events I have described as occurring in the breast are identical in time and sequence with those occurring in the skins of man and mice after the application of tar.

It is a pleasant duty to thank Dr. J. A. Murray, the St. Bartholomew's Hospital Surgical Clinic, Mr. Percy Legg, and Dr. W. R. Smith for the valuable and interesting material thay have placed in my hands. At the same time, it is only fair to these gentlemen to state that they are not responsible for the conclusions I have reached. Finally, I must thank Mr. F. F. Burghard and Dr. May Cutler for making clear parts of this article which appeared obscure.

FURTHER RESULTS OF NERVE ANASTOMOSIS.*

AN ILLUSTRATED RECORD OF SOME EXPERIMENTS IN WHICH:—

1. THE CENTRAL AND PERIPHERAL ENDS OF A DIVIDED NERVE WERE IMPLANTED AT VARYING DISTANCES APART INTO A NEIGHBOURING NORMAL NERVE.

2. CERTAIN NERVE-TRUNKS OF THE LIMBS WERE DIVIDED AND ANASTOMOSED BY SUTURE IN CROSS-WISE FASHION.

By SIR CHARLES BALLANCE,

LIONEL COLLEDGE, and LIONEL BAILEY, London.

The finest sutures and needles have been employed in all the experiments. Sutures used have been Van Horn arterial silk, and linen thread and catgut of about the same size. It does not appear that there is, on examination of microscopical sections, any advantage gained in preferring one kind of material for suture to another. All sutures are dead foreign material, and as such are absorbed. The initial cellular proliferation which ultimately encapsulates and absorbs the suture appears to be much the same in amount whatever material is employed, provided the suture is of the same size; the only difference consists in the time taken in the process of substitution and absorption, i.e., the time required for the replacement of the suture material by living tissue to be complete.

An attempt has been made in each experimental anastomosis to pass the needle and thread only through the neurilemma, but the nerves experimented with are so minute that this attempt must have failed in many instances. The results obtained in these experiments seem to indicate that the passage of the needle and suture only through the neurilemma, though theoretically desirable, is not essential to success in nerve suture.

It has sometimes been said that, in suturing a large nerve-trunk of a limb end to end, corresponding nerve-fibres should be brought into contact. This is impossible in any case: the suggestion is theoretical and is of no practical importance. But on looking at the transverse section of a nerve-trunk it is seen to be made up of bundles of nerve-fibres, some larger than others. The anatomical arrangement of nerve bundles does make it possible in some cases when suturing large nerves end to end to bring corresponding nerve bundles in apposition; but this is impracticable when the minute nerves of the neck of the monkey are the subject of experiment.

Sir David Ferrier, who has taken much interest in these experiments, was anxious that nerve-ends should be brought together and fixed in position without the aid of sutures. For this purpose various varnishes were tried,

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* *See* "Some Results of Nerve Anastomosis," British Journal of Surgery, Vol. xi, p. 327. The Medical Research Council defrayed the expenses of this research, and gave me every assistance in the performance of the experiments, which were carried out at the National Research Institute, Hampstead.—C. B.

such as sandarach varnish, benzo-mastiche varnish, and acetone collodion. It is obvious that no particle of a drop of these varnishes could be allowed to come in contact with the cut end of a nerve, and their use has been given up. The discovery of a living muscle varnish has, however, been successful. The varnish is made by cutting living muscle into small pieces, adding a few drops of sterile salt solution, and pounding the mixture in a sterile mortar with a pestle till a sticky mass is formed. Presumably the sarcolemma is ruptured and the sarcous material within is set free.

In double lateral implantation of one nerve into another, the time required for functional recovery varies with the distance apart of the implanted ends of the nerve, and possibly also with the method of implantation employed. The greatest distance apart of the ends of the external popliteal nerve when implanted into the internal popliteal nerve has been in these experiments 5 cm. But the distance apart of the sites of implantation of one end of the musculospiral nerve into the median nerve at the lower margin of the axilla, and of the other end of the musculospiral nerve into the median nerve just above the bend of the elbow, is much greater than 5 cm. In a small rhesus monkey in which this experiment (not yet complete) was carried out, the distance apart of the two ends of the musculospiral nerve when implanted into the median nerve was fully $8\frac{1}{2}$ cm. ($3\frac{3}{8}$ in.). Fifteen months after the experiment was performed all the muscles of the forearm contracted to a medium faradic current.

In man, massage and electricity are available to maintain the nutrition of the muscles, and the limb is splinted to prevent lengthening of tendons and muscles during the period required for nerve regeneration ; but in monkeys none of these measures is possible. The ankle- and wrist-joints tend to become flexed, and the toes and fingers to curl up, so that it is not true to speak of the recovery that does take place as perfect or complete. In dogs, on the other hand, in which double lateral implantation of the external popliteal nerve into the internal popliteal nerve has been done, the shortening of the flexor muscles and tendons does not take place, as in the act of walking the toes and ankle are by the natural movements extended. In a greyhound, after ten months had elapsed from the date of operation, in which the two ends of the external popliteal nerve were attached to the internal popliteal nerve 5 cm. apart, a median faradic current produced strong contraction in the tibialis anticus, with extension of the ankle, but the toes were only weakly extended. The greyhound could gallop fast over the ground at this date without any apparent disability. The extension of the toes in the dog is normally a weak movement, and the greyhound in galloping and jumping is dependent chiefly on the strong action of the flexors of the ankles and knees of the hind limb. The plan adopted of attaching the ends of the external popliteal to the internal popliteal nerve in this experiment is shown in *Fig.* 356, C.

When an opportunity offered for stimulating directly the nerves in double lateral implantation after functional recovery and before the death of the animal, a notable result was obtained. On faradic stimulation of the peripheral segments of the two nerves it was found that a stronger current was required to produce the same effect than was necessary when faradic

stimulation was applied to the central segments of the two nerves—in other words, the same strength of current caused less and less response as the stimulation proceeded from the central to the peripheral parts of the nerves.

This phenomenon has been described as the phenomenon of the avalanche (*see* G. Durante's article in Cornil and Ranvier's *Manuel d'Histologie pathologique*, 3rd edition, 1907, Vol. III, p. 495 : " Phénomène de l'Avalanche (Pfluger), de la Boule de Neige (Chauveau) "). Durante points out, on p. 603, that functional restoration may occur (" transmission active de neuroblaste à neuroblaste : conductibilité plasmique ") while there is still incomplete regeneration of the nerve-fibre, and that it is important to distinguish this function of conductivity from that of excitability. He describes the latter as follows : " pouvoir de transformer des vibrations (excitations) diverses en des vibrations nerveuses ". There is in the above experiment a clear differentiation between the phenomenon of conductivity and that of excitability. The difference in functional activity of the nerve-fibres, in the central and peripheral segments of the nerves, appears to depend on a difference in histological or anatomical structure—in other words, on the greater or less perfection of the process of regeneration attained by them.

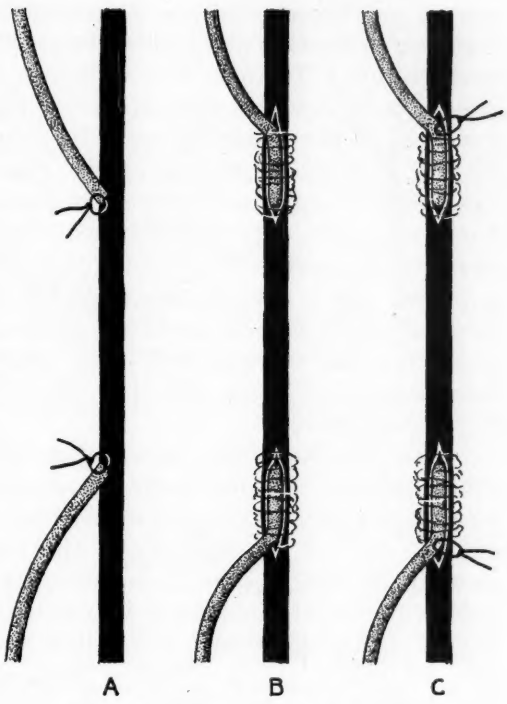


FIG. 356.—Diagrams of the methods employed in double lateral implantation for fixing the ends of a divided nerve to a neighbouring nerve. A, Two transverse incisions were made in the side of the normal nerve for about one-third of its diameter. The ends of the divided nerve were fixed into the incisions by one suture of iron-dyed silk, linen thread, or catgut. The sutures were the finest obtainable (arterial size) and were threaded on the finest needles. The attempt was always made to suture sheath to sheath, but it is not claimed that this was always accomplished. B, Implantation of the ends of the divided nerve into median incisions made in the normal nerve. Fixation was effected by ' muscle-varnish ' without sutures. C, Implantation of the ends of the divided nerve into median incisions made in the normal nerve. Fixation was effected by ' muscle-varnish ' and by one suture ; each suture passed through the sheaths of both nerves half an inch from the end of the divided nerve.

It remains to discuss the best method of attaching the two ends of a divided nerve in double lateral implantation to a neighbouring nerve (*Fig.* 356). Is it better to make transverse incisions, at a certain distance apart, in the course of the nerve into which the two ends of the implanted nerve are to be sutured ; or is it better to implant the two ends into median incisions ? At this stage of the experiments it does not seem possible to come to any conclusion. It would seem, however, that when median incisions are made in the receiving nerve, there is a tendency, in the case of double lateral implantation of the external popliteal

nerve into the internal popliteal nerve, for the two ends to become connected with fibres originally belonging to both the external and the internal popliteal nerves, so that on proximal stimulation of either nerve the response may be, not a pure flexor or extensor one, but mixed flexor and extensor, one movement, however, being more marked than the other. When incisions are made in the side of the normal nerve, a portion of it is cut off from its previous connections both distally and proximally. This isolated band of nerve-fibres, together with the connective tissue of the sheath, is the site of a cellular proliferation. There is thus formed a new tissue through which nerve-fibres penetrate or develop, and ultimately produce a functional union between the two ends of the divided nerve. In such an experiment, on faradic stimulation of the proximal segments of the two nerves, pure effects of flexion and extension of ankle and toes have been observed. But this is probably not true of all such experiments, for on examination of some of the microscopical drawings the nerve-fibres of the central and peripheral ends of the external popliteal nerve are clearly connected with nerve-fibres belonging to both internal and external popliteal nerves. Nerve-fibres have a spiral arrangement in a nerve-trunk, and hence what appears to be clearly exhibited in a microscopical drawing may not indicate the true functional connections of the nerve-fibres.

In the notes of the experiments, the dates on which faradic stimulation was applied to the previously paralysed muscles are given. It is desirable to remember that this date, if a response to the electrical stimulation occurs, is not the date of the recovery of the muscle. The frequent etherization of a monkey for the purpose of determining the earliest date at which a response follows faradic stimulation is impossible. The risk to the animal, and possible failure of the experiment, which in any event requires that the monkey should live for many months, makes it essential that an anæsthetic should only occasionally be administered.

The microscopical sections have been cut, stained, and mounted by Mr. Steward at the Royal College of Surgeons, by the kind permission of Sir Arthur Keith.

DETAILS OF EXPERIMENTS.

Experiment 1.—**Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.**

Rhesus Monkey.—External popliteal nerve divided and attached in two places to incisions made in the side of the internal popliteal nerve ½ in. apart; one suture used for each anastomosis, of iron-dyed silk (Van Horn). Operation on Dec. 5, 1922.

Jan. 17, 1923.—Foot-drop marked.

April 26.—Climbs well with both feet.

May 23.—*Faradic stimulation.* Definite contraction (slow response) in tibialis anticus and peronei muscles; none in extensor longus digitorum or extensor hallucis. Flexors of ankle contract almost normally.

July 19.—*Faradic stimulation* (6½ *months*). Extensor communis digitorum contracts. It is not possible in the monkey to keep the ankle and toes extended during the period of nerve regeneration, hence the toes are some what curled up in walking.

Oct. 23.—*Faradic stimulation.* All the muscles of the foot and toes supplied by the external popliteal nerve contract, but they all require a somewhat stronger current than the corresponding muscles of the other or normal limb. Galvanic response normal.

Nov. 20.—11½ *months.* The animal was ill, and hence was killed by ether inhalation. Before death occurred the anastomoses were exposed and stimulated with the faradic current. (Sir David Ferrier was present.)

Results of Stimulation (*Fig.* 357) :—

B and D, Flexion of ankle and toes.
A and C, Extension of ankle and toes.
E, Extension of ankle and toes.
F, Flexion of ankle and toes.

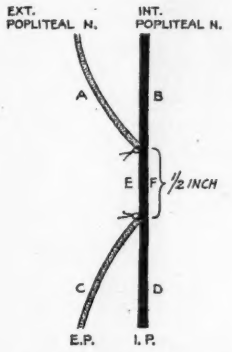


FIG. 357.—*Experiment* 1. Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 11½ months.

Microscopical Appearances.—The proximal segment of the external popliteal nerve ends in a bulb (*Fig.* 358), and from it nerve sheaths extend downwards, apparently in new tissue on the outer aspect of the internal popliteal nerve-trunk between the two anastomoses.

The distal segment of the external popliteal nerve (*Fig.* 359) exhibits complete regeneration, its nerve-fibres being in direct communication with those of the proximal segment in the tissue on the outer side of the internal popliteal nerve-trunk. The activity of regeneration appears to be not less in the lower segment than in the upper segment.

Proximal to the bulb the main nerve-trunk stains deeply, as does also the whole length of the uninjured internal popliteal nerve. In the bulb the sheaths are finer, very numerous, and lightly stained. They interlace in many directions, but mainly tend to run longitudinally. At the level of the bulb there is a place on the outer side of the internal popliteal nerve where regeneration has taken place, and here also the neurilemma sheaths are fine, numerous, and not deeply stained. The same is true of the region of the distal anastomoses. The sheaths stain lightly, they interlace, and are numerous. Beyond the anastomosis area the sheaths of the main trunk of the regenerated distal segment of the external popliteal nerve stain deeply. The new tissue between the proximal and distal segments of the external popliteal nerve is permeated by numerous groups of stained sheaths running in a longitudinal direction. They do not seem to have any connection with the internal popliteal nerve. Wherever regeneration has taken place are seen numerous neurilemma cells with rod-shaped nuclei. These lie alongside the new sheaths. Connective-tissue cells with oval nuclei are present also.

Before the death of the monkey the upper part of the opposite Rolandic area was stimulated. Extension of the ankle and toes occurred at a spot a little above the area where stimulation caused flexion of the ankle and toes. These results of stimulation of the cortex were like those obtained by stimulating the corresponding areas of the opposite Rolandic cortex.

Experiment 2.—**Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.**

CAT.—External popliteal nerve divided and the ends implanted in two places into the internal popliteal nerve, 4 cm. apart. The ends of the external popliteal nerve were inveigled into longitudinal slits made in the middle of the internal popliteal nerve. The anastomoses were fixed in position with 'muscle varnish'. An inch from each anastomosis two drops of 'benzo-mastiche' varnish were applied to each segment of the external popliteal nerve. This varnish fixed the proximal and distal parts of the external popliteal nerve to neighbouring muscular tissue. It was hoped in this way to secure the anastomoses from displacement, as no sutures were used.

2 *months and* 20 *days.* The animal suffered from diarrhœa and was killed by ether inhalation.

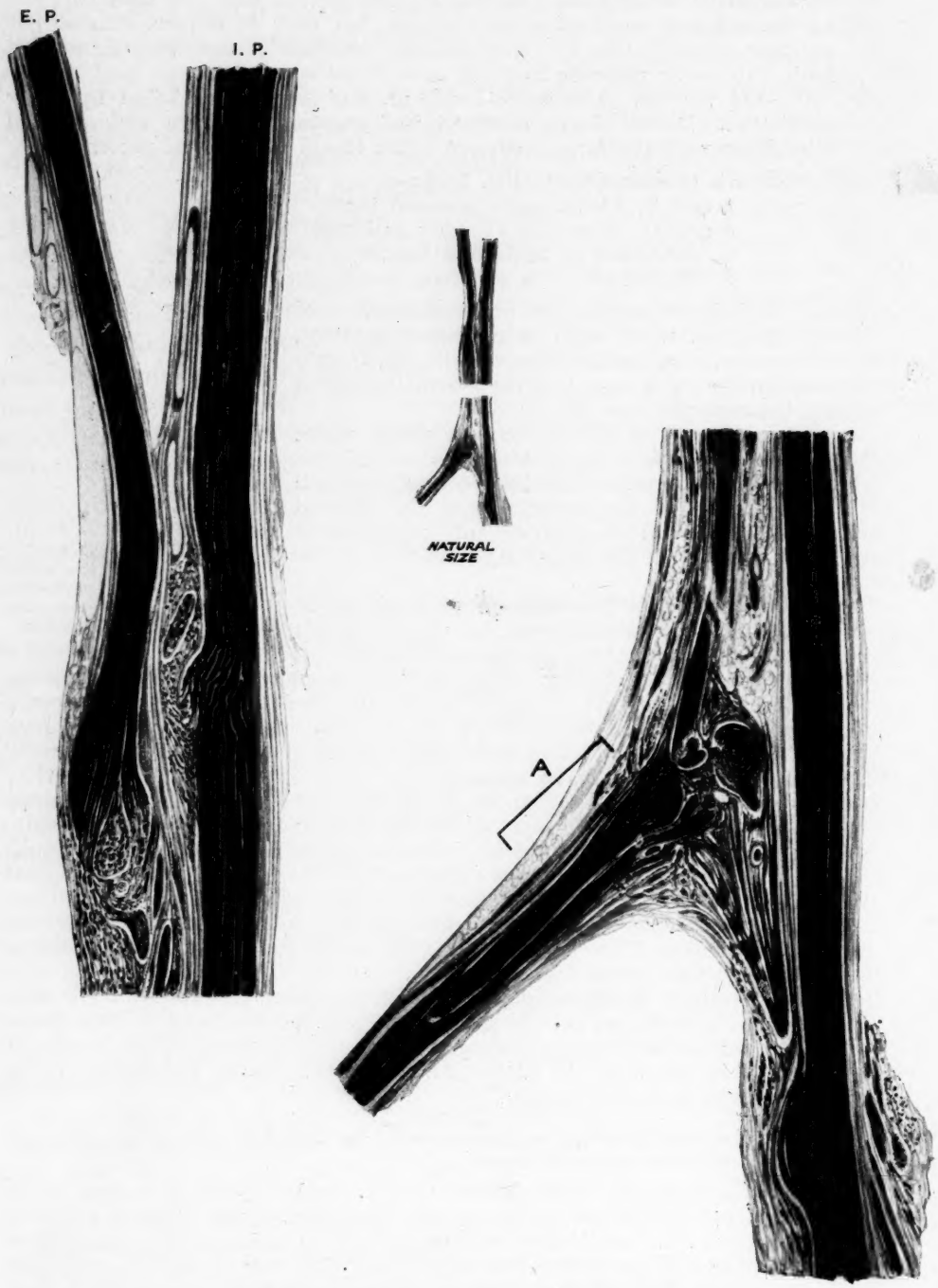


FIG. 358.—*Experiment* 1. Monkey. External popliteal nerve divided and the ends anastomosed in two places, half an inch apart, to the side of the internal popliteal nerve. Specimen obtained 11½ months afterwards. Weigert stain. I. P., Internal popliteal nerve; E. P., External popliteal nerve; A, Portion shown more highly magnified in *Fig.* 359. (× 7.)

FIG. 359.—Distal part of external popliteal nerve marked A in *Fig.* 358. (× 42.)

FIG. 360.—*Experiment* 2. Cat. Double lateral implantation of external popliteal nerve into two incisions, 4 cm. apart, made in the middle of the internal popliteal nerve. Specimen obtained 2 months and 20 days afterwards. Note reproduction of nerve-fibres in distal segment of external popliteal. Cajal stain. (×7.) A is a more highly magnified (× 460) portion of the distal segment of the external popliteal shown at X.

Microscopical Appearances.—In the low-power drawing (*Fig.* 360) it is seen that the distal end of the proximal part of the external popliteal nerve ends in a bulb, some of the fibres of which join fibres of the internal popliteal nerve, while others pass into the connective tissue on the outer side, in which are other columns of nerve-fibres.

The distal segment of the external popliteal shows marked regeneration of nerve-fibres. The proximal end of the distal segment spreads out in a wide bush of fibres which spread upwards to join nerve columns from the outer to the inner border of the internal popliteal nerve. These newly regenerated fibres are finer than those which are seen in undamaged nerve-trunks (*Fig.* 360, A).

It would appear from the position of the two ends of the external popliteal nerve that both had shifted after the operation: the ends of the proximal segment upwards from its position in the proximal median incision in the internal popliteal nerve, and the end of the distal segment downwards from its original position in the distal median incision in the internal popliteal nerve.

Experiment 3.—**Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.**

RHESUS MONKEY.—External popliteal nerve divided and ¾ in. of the nerve cut away. Ends of external popliteal nerve implanted into incisions made in the side of the internal popliteal nerve, 1 in. apart. Two sutures of iron-dyed arterial silk employed to fix each anastomosis.

4 *months.*—No response to faradic current in extensors of ankle and toes.

7 *months.*—Response to stimulation with faradic current: Tibialis anticus and extensor communis digitorum. (Sir David Ferrier present.)

10½ *months.*—Toes still curled up. Ankle cannot be extended beyond a right angle because of shortening of flexor tendons and muscles. Animal was ill, hence was killed by ether inhalation.

Before death the muscles were stimulated with the faradic current through the skin: *All the extensor* muscles of the ankle and toes responded to a medium current, also the peronei muscles; the flexors also all contracted.

The anastomoses and nerves were exposed and stimulated. A weak faradic current was used.

Results of Stimulation (*Fig.* 361):—

A, B, and C, Contraction of all the extensors and the peronei.

D, E, and F, Contraction of all flexors of ankles and toes.

On stimulating A and D the strongest movement was exhibited.

,, ,, C and F the movement was markedly less.

,, ,, B and E the weakest movement of either flexion or extension was observed.

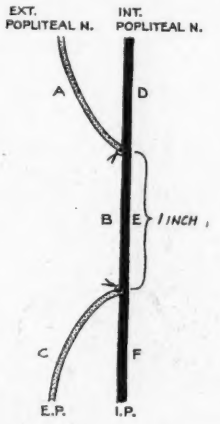


FIG. 361.—*Experiment* 3. Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 10½ months.

Microscopical Appearances.—We have no record of the distal anastomosis as this was unfortunately lost.

The proximal anastomosis is shown in *Fig.* 362. The site of the incision in the internal popliteal nerve is obvious; it exhibits a plexus of nerve-fibres spreading in many directions.

Below the anastomosis the external popliteal nerve is continued in the new tissue formed external to the uninjured part of the internal popliteal nerve, though some fibres appear to join those of the internal popliteal nerve at the site of the anastomosis.

Note reproduction of nerve-fibres in distal segment of external popliteal. Cajal stain. (× 7.) A is a more highly magnified (× 450) portion of the distal segment of the external popliteal shown at X. afterwards. I. P.

FIG. 362.

Experiment 4.—**Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.**

CAT.—External popliteal nerve divided and the ends implanted into incisions made in the middle of the internal popliteal nerve. The ends of the external popliteal nerve were 3 cm. apart. The incisions in the internal popliteal nerve appeared to be more like separations of nerve bundles than divisions of nerve-fibres: it was doubtful if nerve-fibres were cut. The anastomoses were kept in place by 'muscle varnish', made by pounding in a mortar a small piece of living muscle with a few drops of sterilized salt solution. This produced a sticky mass which held the nerves together. After a few minutes the 'varnish' was adherent and could be pulled on by forceps without coming away. This was the first occasion on which 'muscle varnish' was used.

FIG. 362.—*Experiment* 3. Monkey. Double lateral implantation of the divided external popliteal nerve into the side of the internal popliteal nerve. The figure shows the upper anastomosis; the lower anastomosis was lost. Specimen obtained 10½ months afterwards. Cajal stain. S, Site of suture. (× 10.)

1½ *months.*—Wound opened; external popliteal nerve-ends in place; a good deal of new tissue, red and vascular, around each anastomosis.

Results of Faradic Stimulation (*Fig.* 363):—

A and B, No extension.
C, Sharp flexion.
D, A little less flexion.
E, Very slight flexion.
The same strength current used in each case.

3 *months.*—No lameness in walking. This is an acrobatic cat; climbs anywhere; there is a wire netting near the ceiling of the room in which the cats were kept. This animal would spring from a ledge to this netting and walk along upside down, there being no appearance of weakness of the operated limb. On holding the cat up by the shoulders there was not so much dorsiflexion of the ankle of the operated limb as of the ankle of the normal limb: but the claws of both limbs were equally exposed. (Sir David Ferrier was present.)

5 months.—Claws and ankle extended strongly.

10 months.—Animal etherized and subsequently killed with ether.

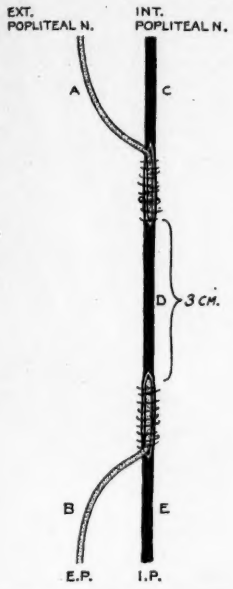


FIG. 363.—*Experiment* 4. Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 1½ months.

Faradic Stimulation through Skin :—

1. At the back of thigh: Flexion of ankle and toes and protrusion of claws (? extension) occurred.
2. Over upper and outer part of leg, over region of external popliteal nerve: Extension of ankle, toes, and claws.

The anastomoses were exposed and the nerves stimulated with the faradic current.

Results of Stimulation (*Fig.* 364) :—

A and B, Extension of ankle, toes, and protrusion of claws.
E, Extension of ankle, toes, and protrusion of claws.
C, Flexion of knee, ankle, toes, and protrusion of claws.
D, Flexion of ankle, toes, and protrusion of claws.
F, Variable: sometimes flexion, sometimes extension, as if internal popliteal nerve-fibres were joined to both nerves distal to this point of stimulation.

At B and D more current was required to produce the same effect than was necessary at A and C.

Microscopical Appearances.—*Fig.* 365 shows the position of the two ends of the external popliteal nerve within the internal popliteal nerve. *Fig.* 365, C shows the regeneration of fibres in the distal segment of the external popliteal nerve. *Fig.* 366, A is an enlarged view of the bulb at the end of the proximal segment, and shows the multitude of new fibres present. *Fig.* 366, B is an enlarged picture of the proximal end of the distal segment. Without examining serial sections it is not easy to follow the anatomical connections of the two ends of the external popliteal nerve with the columns of fibres on either side and beyond them.

Experiment 5.—**Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.**

CAT.—External popliteal nerve divided and the ends united to transverse incisions made in the side of the internal popliteal nerve, 4½ cm. apart.

4½ months.—Animal walking well. The toes were slightly flexed when at rest as compared with those on the normal side. Both extension and flexion of the ankle were strong movements.

At this date the animal was killed by ether inhalation, but before death occurred the anastomoses were exposed and stimulated with the faradic current. (Sir David Ferrier was present.)

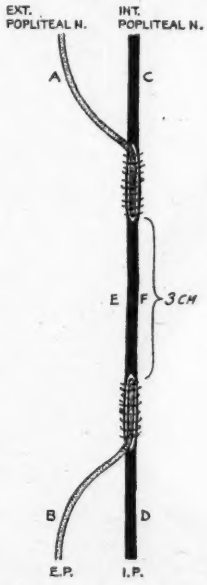


FIG. 364.—*Experiment* 4. Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 10 months.

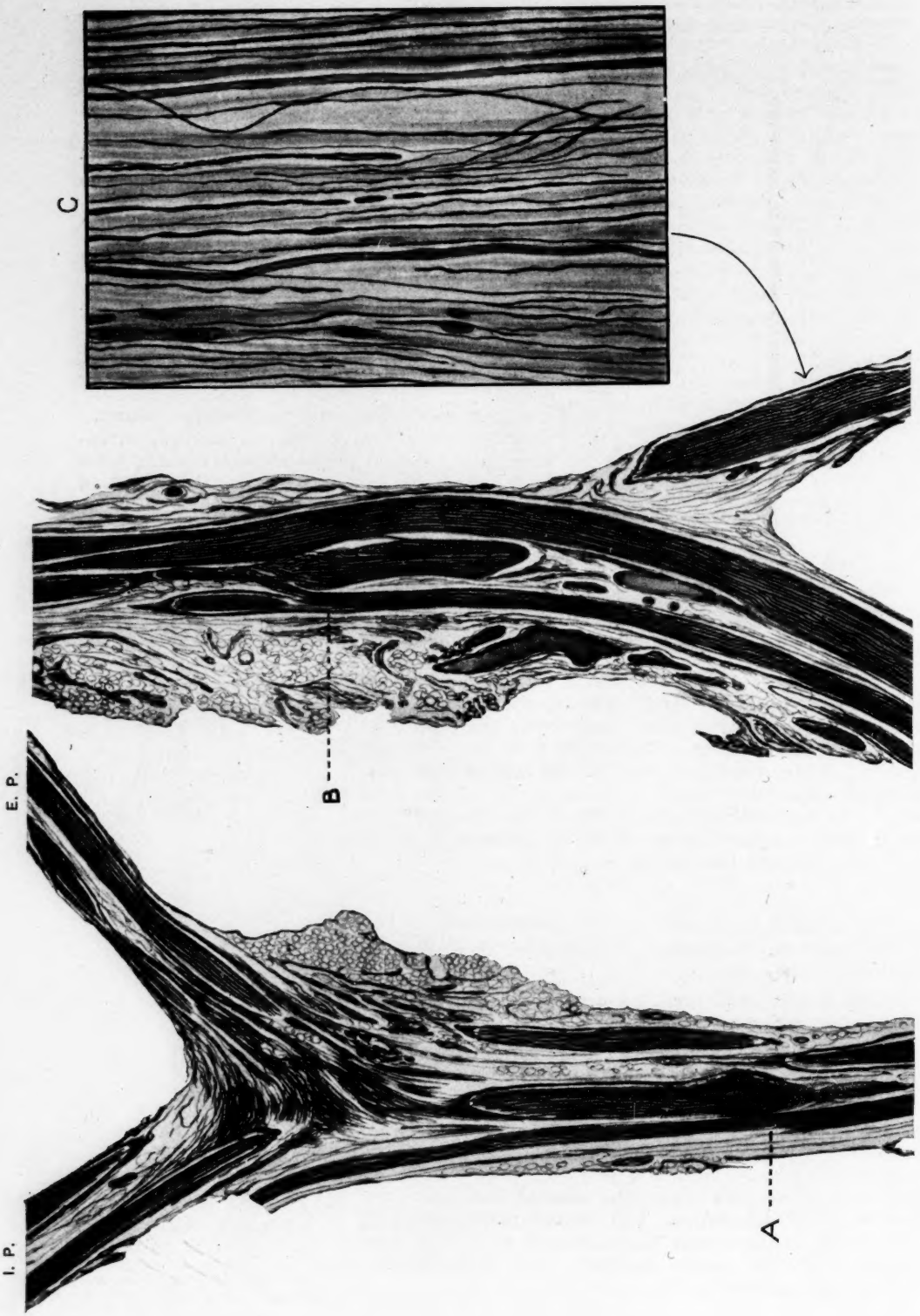


FIG. 365.—*Experiment* 4. Cat. . External popliteal nerve divided and the ends implanted into two incisions made in the middle of the internal popliteal nerve, 3 cm. apart. Specimen obtained 10 months afterwards. (× 7.) C is a more highly magnified (× 450) portion of the distal segment of the external popliteal nerve to show the regeneration of fibres. A and B indicate portions more highly magnified in *Fig.* 366.

A

B

FIG. 366.—A, Bulb (somewhat compressed) of distal end of proximal segment of external popliteal nerve shown at A in *Fig.* 365. (× 100.) B, Proximal end of distal segment of external popliteal nerve shown at B in *Fig.* 365. (× 45.)

internal popliteal nerve, 3 cm. apart. Specimen obtained 10 months afterwards. (× 7.) C is a more highly magnified (× 450) portion of the distal segment of the external popliteal nerve to show the regeneration of fibres. A and B indicate portions more highly magnified in *Fig.* 366.

Results of Stimulation (*Fig.* 367) :—

A, Extension of ankle (strong) and marked protrusion of claws.
B, Flexion of ankle ; with full flexion claws were exposed.
C, Flexion of toes. Flexion of ankle, but not such a strong movement as at B.
D, Flexion of ankle. Strong flexion of toes ; claws not protruded.
E, Strong flexion of ankle and slight flexion of toes.

The transverse incisions in the internal popliteal nerve extended across the nerve for half its diameter. It would seem that the upper segment of the external popliteal nerve was at this stage of the experiment (4½ months) chiefly connected with fibres of the internal popliteal nerve. The flexion movement is the stronger, and it might have masked a weak extension movement when E was stimulated : or perhaps it was too soon for functional union to have taken place between the proximal and distal segments of the external popliteal nerve. The striking fact was that when A (distal segment of external popliteal nerve) was stimulated, strong extension of ankle and toes occurred, so this part of the external popliteal nerve had reached the stage of regeneration in which it exhibited the phenomenon of excitability.

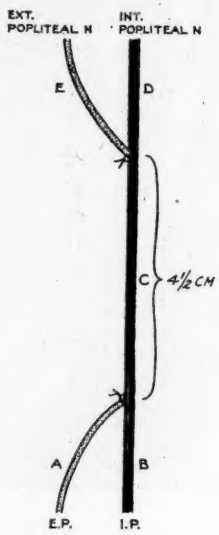


FIG. 367.—*Experiment* 5. Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 4½ months.

Experiment 6.—**Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.**

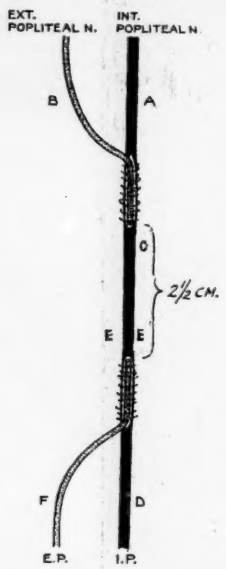


FIG. 368.—*Experiment* 6. Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 9 months.

CAT.—External popliteal nerve divided and the ends inveigled into longitudinal median incisions in the internal popliteal nerve, 2½ cm. apart. The anastomoses were fixed with 'muscle varnish'.

3 *months.*—Walks well. On being held up by the shoulders the ankle is extended and the claws protruded, but not so much as on normal side.
7 *months.*—Ankle extended and claws protruded as on normal side.
9 *months.*—Animal was ill, and was killed by ether inhalation. Before death the nerves were exposed and stimulated with the faradic current.

Results of Stimulation (*Fig.* 368) :—

A, Strong flexion of toes.
B, Combined movement. Contraction both of extensor and flexor muscles.
C, Less strong flexion of toes.
D, Only slight flexion of toes.
E, E, Extension of ankle and toes.
F, " " " " " but not so strongly marked as at E, E.

Experiment 7.—**Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.**

CAT.—External popliteal nerve divided and the ends inveigled into longitudinal median incisions made in the internal popliteal nerve, 3 cm. apart. The anastomoses were fixed by 'muscle varnish'.

3 *months.*—Only slight extension of ankle and toes on being held up by the shoulders.

At this date the animal was ill, and was killed by ether anæsthesia. Before death the nerves were exposed and stimulated with the faradic current. (Stimulation of the extensor muscles through the skin produced no response.)

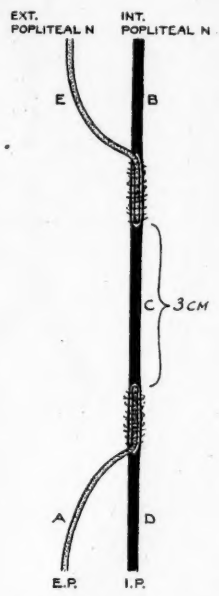


FIG. 369.—*Experiment* 7. Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 3 months.

Results of Direct Stimulation (*Fig.* 369) :—

A, Extension of ankle and toes.
B, Strong flexion of ankle and toes.
C, Less strong flexion of ankle and toes.
D, Only slight flexion of ankle and toes.
E, Combined flexion and extension movements : the flexion movement being the most powerful.

Experiment 8.—**Double lateral anastomoses of the divided ends of the ulnar nerve to the median nerve.**

RHESUS MONKEY.—Double lateral implantation of divided ulnar nerve into the side of the median nerve, $1\frac{1}{8}$ in. apart. The median nerve was incised for $\frac{1}{3}$ in. (or more) at the site of each anastomosis. The ends of the divided ulnar nerve were passed under the superficial muscles and tendons of the forearm to reach the anatomical position of the median nerve. One suture of Van Horn arterial iron-dyed silk was used to fix each anastomosis. The attempt was made to pass each suture through the sheath of the nerves only. The median and ulnar nerves (distal portions) were stimulated with the faradic current before the anastomoses were carried out. These stimulations caused contraction of the intrinsic muscles of the hand supplied by each nerve. The most striking movements were the flexion of the metacarpophalangeal joints by the lumbricales muscles and the drawing inwards of the thumb over the palm by the adductor and the opponens pollicis muscles.

4 *months.—Faradic stimulation.* Response is present in all the intrinsic muscles supplied by the median nerve. The intrinsic muscles supplied by the ulnar nerve—the interossei, the two inner lumbricals, the muscles of the hypothenar eminence, and the deep intrinsic muscles of the thumb—all contract, but the response to stimulation is not so easily obtained as is the response of the intrinsic muscles supplied by the median nerve.

5 *months.—Faradic stimulation.* Response of all the intrinsic muscles of the hand obtained with more ease. The hand is being used for all purposes, but when food is offered the undamaged hand takes it.

$8\frac{1}{2}$ *months.* — Response to galvanic current in all intrinsics of hand normal. Response to weak normal faradic current is easily obtained in all the muscles.

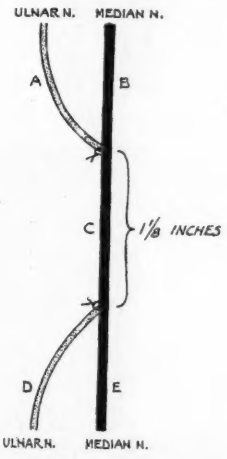
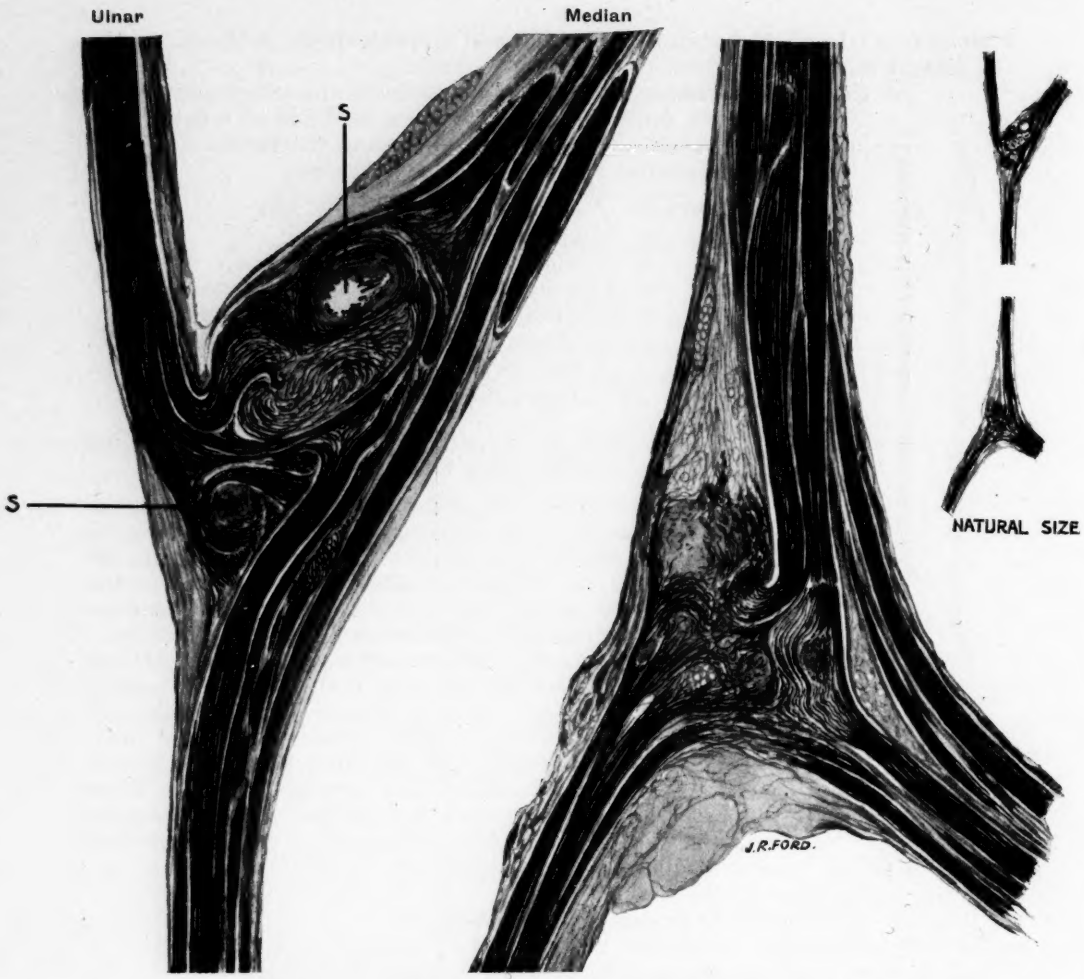


FIG. 370.—*Experiment* 8. Diagram showing the points on the ulnar and median nerves at which faradic stimulation was applied. 10 months and 10 days.

10 *months and* 10 *days.*—The monkey was killed by ether inhalation. Before death the anastomoses were exposed and the nerves stimulated with the faradic current.



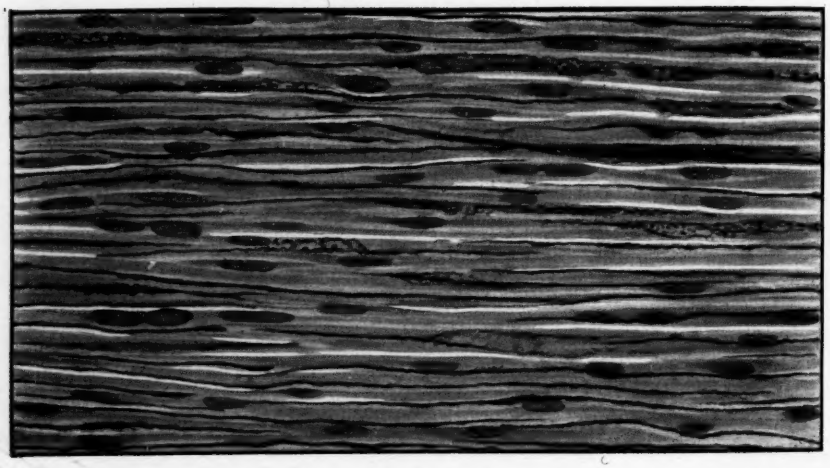


FIG. 371.—*Experiment* 8. Monkey. Double lateral implantation of divided ulnar nerve into the side of the median nerve, $1\frac{1}{2}$ in. apart. Specimen obtained 10 months and 10 days afterwards. Weigert stain. S, Site of suture. (× 7.) A, High-power view of distal part of ulnar nerve. Stroebe stain. (× 450.)

Results of Stimulation (*Fig.* 370) :—

A, Response of median intrinsics.
B, ,, ,, ulnar ,,
C, ,, ,, both median and ulnar intrinsics.
D, ,, ,, median intrinsics, weaker movement than at A (same current).
E, ,, ,, ulnar intrinsics, weaker movement than at B (same current).

Microscopical Appearances.—The microscopical specimens were stained by the Weigert and Stroebe methods. In *Fig.* 371 the appearances show that the median nerve was considerably injured by the transverse incision made at the operation. There is no new tissue on the ulnar side of the main trunk of the median nerve, as is seen in a corresponding position when this type of operation is performed on the popliteal nerves ; but in some of the sections this tissue is visible and contains columns of new nerve-fibres. S shows the site of the remains of one of the sutures. *Fig.* 371, A is a high-power view of the regenerated distal segment of the ulnar nerve (the Stroebe stain showing the new axis cylinders). The new fibres joining the ends of the ulnar nerve to the median nerve are very numerous, and group themselves in various curving bands of fibres which appear definitely to join with corresponding groups of median nerve-fibres. The right forearm of the animal was operated on, but, in the process of mounting, the sections were turned over so that in the figure the nerves are placed as in the left forearm. The lateral spreading out of the bands of fibres of which the median nerve is composed is due to the tension on the tissues of the nerve when pinned out on a sheet of cork during the hardening process.

Experiment 9.—**Double lateral anastomoses of the divided ends of the median nerve to the ulnar nerve.**

Rhesus Monkey.—Double lateral implantation of divided median nerve of forearm into the side of the ulnar nerve, 1¼ in. apart. The ulnar nerve at the site of the proximal anastomosis was divided almost completely, while at the site of the distal anastomosis about one-half of the nerve was divided. The ends of the median nerve were passed under the superficial muscles and tendons of the forearm to reach the anatomical position of the ulnar nerve. One suture of Van Horn arterial iron-dyed silk was used to fix each anastomosis. The natural tension of the living nerve caused the incisions in the ulnar nerve to gape considerably.

Before the experiment was commenced both nerves were stimulated with the faradic current. *Median nerve stimulation* : Flexion of the metacarpal phalangeal joints of the 1st and 2nd fingers, and the thumb was drawn across the palm (opponens pollicis). *Ulnar nerve stimulation* : Flexion of 3rd and 4th fingers at the metacarpal phalangeal joints, and adduction of thumb. The other intrinsic muscles of the hand contracted, but the above movements were the most important visible effects of stimulation of these two nerves.

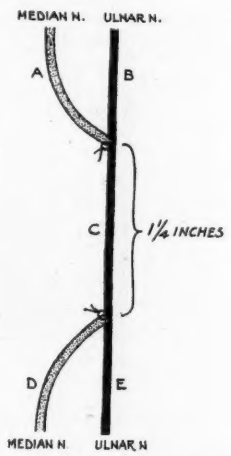


Fig. 372.—*Experiment* 9. Diagram showing the points on the median and ulnar nerves at which faradic stimulation was applied. 10½ months.

4½ *months.*—*Faradic stimulation of muscles.* Response observed in all the intrinsic muscles of the hand, including the two outer lumbricals.

5½ *months.*—Same report.

9 *months.*—*Faradic stimulation.* All the median intrinsics respond to a weak current.

10½ *months.*—Monkey was ill, and was killed by ether inhalation. Before death the anastomoses were exposed and the nerves stimulated with the faradic current.

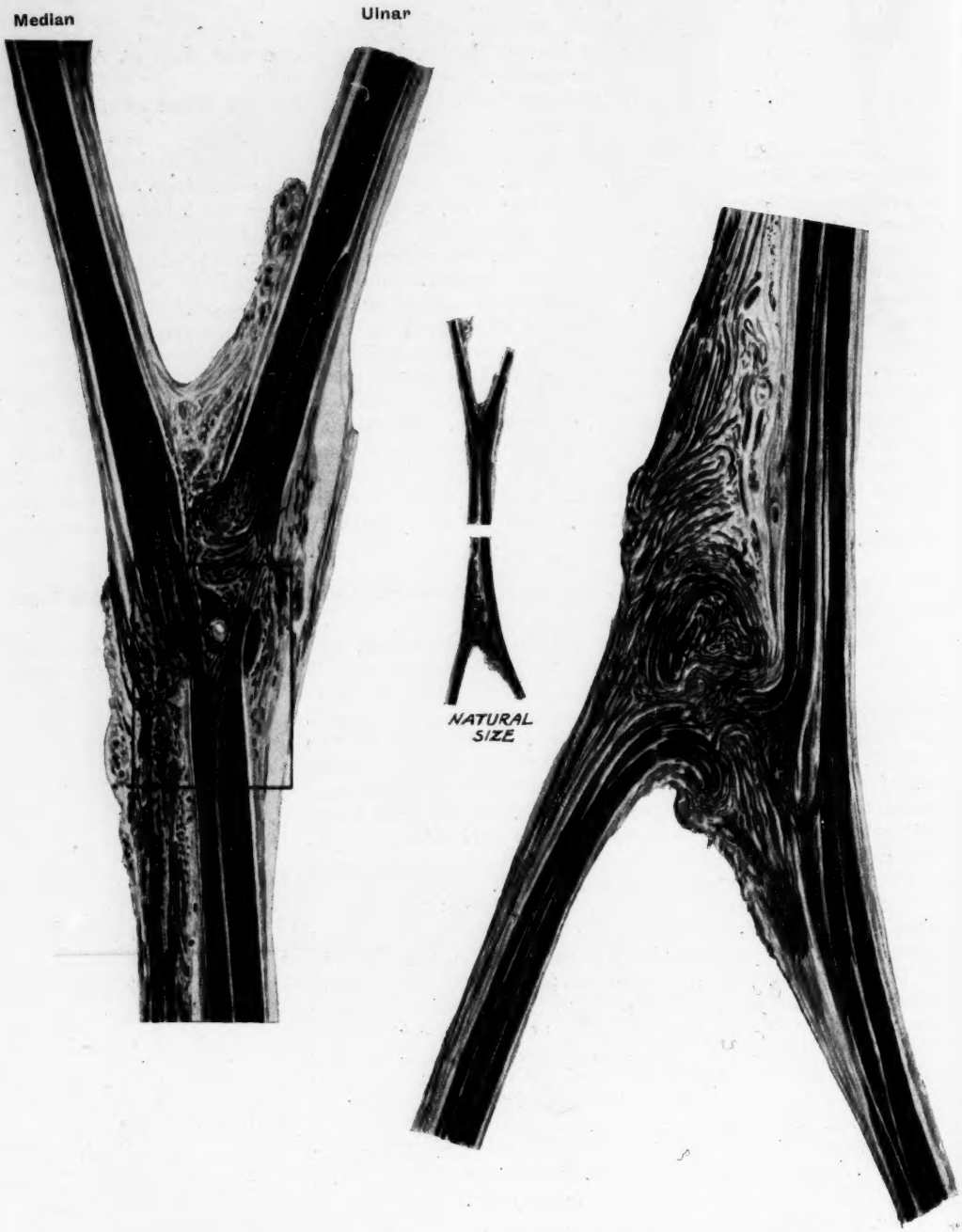


FIG. 373.—*Experiment* 9. Monkey. Double lateral implantation of divided median nerve of forearm into the side of the ulnar nerve, 1¼ in. apart. Specimen obtained 10½ months afterwards. Weigert stain. The black rectangle shows the part included in *Fig.* 374, A. (× 7.)

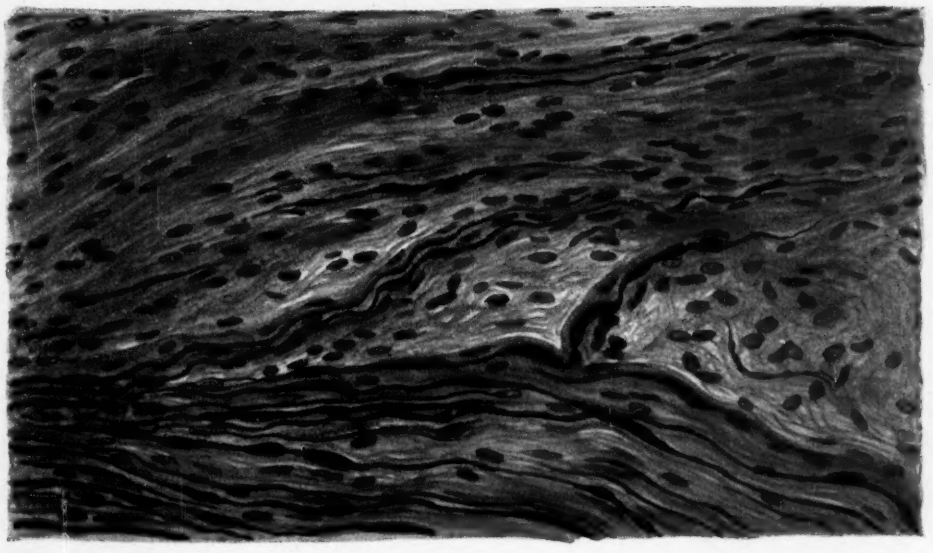


FIG. 374.—A, More highly magnified view of part included in black rectangle in *Fig.* 373. Upper anastomosis, showing fibres of ulnar nerve continuous with median nerve at site of suture. 1, New median nerve-fibres external to main ulnar trunk; 2, Part of ulnar nerve continuous at site of suture with both median and ulnar fibres; 3, The other half of ulnar nerve not communicating with median nerve. (× 30.) B, High-power view of the part included in the black rectangle in *Fig.* 375, 2. Stroebe stain. (× 300.)

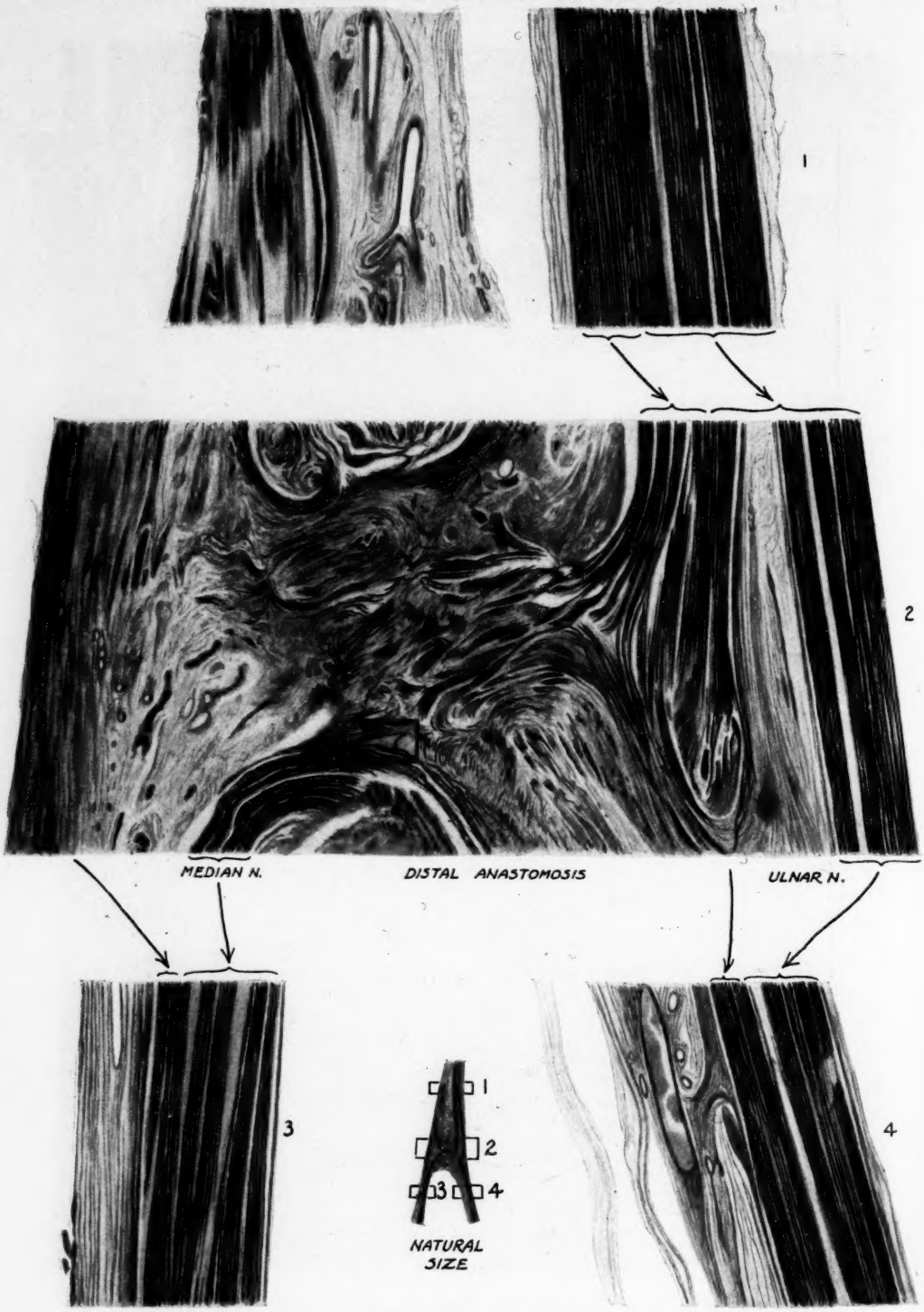


FIG. 375.—Higher-power view of the parts in the neighbourhood of the distal anastomosis shown in *Fig.* 373. 1 shows, on the right, part of the ulnar nerve above the anastomosis isolated by incisions into which the divided ends of the median nerve were sutured; this part has regenerated, and through it impulses pass from the proximal to the distal portions of the median nerve; on the left is seen the tissue external to the nerve between the two anastomoses. 2 shows the distal anastomosis itself; the part included in the black rectangle is shown under higher magnification in *Fig.* 374, B. 3 and 4 show the median and ulnar nerves respectively below the anastomosis. Stroebe stain. (× 30.)

Results of Stimulation (*Fig.* 372) :—

A, Response of median intrinsics.	}	Weak current.
B, ,, ,, ulnar ,,		
D, ,, ,, median intrinsics	}	Current stronger than required at A and B.
E, ,, ,, ulnar ,,		
C, No response.		

Microscopical Appearances.—*Fig.* 373 gives a general view of the anastomosis.

Fig. 374, A is an enlarged view of the proximal anastomosis. The part selected for the drawing is marked by lines in *Fig.* 373. The course of the fibres at the anastomosis is indicated in the legend below the drawing. The wide band of new tissue on the median side of the ulnar nerve, occupying the region between the two transverse incisions which were made in the side of the ulnar nerve at the operation, is seen to be traversed by new sheaths which are connected with the proximal end of the median nerve.

Fig. 375 includes sections stained by the Stroebe method to show the distal anastomosis, the median and ulnar nerves above and below the distal anastomosis, and the nerve-fibre anastomoses. It would appear that the nerve-fibres of the distal segment of the median nerve have formed three connections : (*a*) With nerve-fibres in the new tissue on the median side of the main ulnar nerve-trunk ; (*b*) With a band of nerve-fibres of the main ulnar nerve-trunk ; and (*c*) With fibres of the ulnar nerve beyond the distal anastomosis.

Fig. 374, B is a high-power drawing of a portion of the drawing marked 2 in *Fig.* 375, stained by the Stroebe method. The part selected for the drawing is marked by lines. The rod-shaped nuclei of numerous neurilemma cells cover the whole field. They lie in the same direction as the developing young axis cylinders. The neurilemma cells are spindle shaped, and form long lines of cells with the processes in contact or overlapping.

The young axis cylinders, when examined with a Zeiss $\frac{1}{12}$ oil immersion lens and ocular 2, appear to lie in the protoplasm of the neurilemma cells. When a Weigert-stained specimen is examined under the same magnification, the young sheaths lie also in the protoplasm of the neurilemma cells. The impression produced is that both axis cylinders and sheaths are developed from the protoplasm of the neurilemma cells.

Experiment 10.—**Double lateral anastomoses of the divided ends of the median nerve to the ulnar nerve.**

Rhesus Monkey.—Median nerve of forearm divided, and the ends, after being passed under the flexor muscles and tendons, inveigled into median incisions made in the trunk of the ulnar nerve, 3 cm. apart. Half an inch from each end of the median nerve a fine silk suture was passed through the sheaths of both median and ulnar nerves (*Fig.* 376). In this manner the fixing sutures were each some distance from the cut ends of the median nerve. No 'muscle varnish' was used.

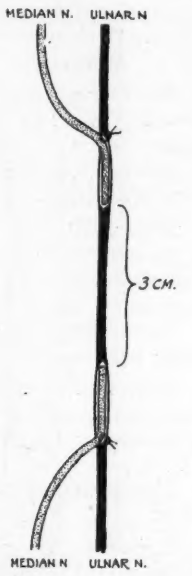


Fig. 376.—*Experiment* 10. Diagram showing ends of divided median nerve inveigled into median incisions in trunk of ulnar nerve.

4 *months.*—*Faradic stimulation.* Moderate strong current : All the median intrinsics contracted, but the 2nd lumbrical showed only a weak movement. Moderate current : Same report. All the ulnar intrinsics contracted, including all the interossei and the 2 inner lumbricals.

8 *months.*—Median and ulnar intrinsics all react to a weak current. A film of the movements was taken.

The experiment is not yet complete.

Experiment 11.—**Crosswise anastomoses of the median and musculospiral nerves.**

BABOON.—The median and musculospiral nerves were divided in the upper arm region. The proximal part of the musculospiral was sutured to the distal segment of the median nerve and the end of the proximal segment of the median nerve was sutured to the distal part of the musculospiral nerve. Two sutures of Van Horn arterial silk were used to fix each anastomosis (*Fig.* 377). A piece of fascia lata was wrapped round each anastomosis so as to prevent the anastomoses coming into contact.

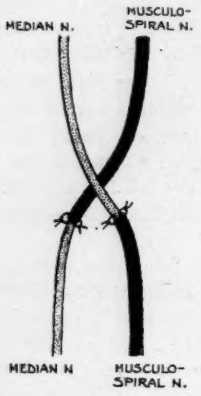


FIG. 377.—*Experiment* 11. Diagram showing crosswise anastomoses operation of the median and musculospiral nerves.

1½ *months.*—*Faradic stimulation.* No movement observed in muscles supplied by the median and musculospiral nerves distal to the anastomoses.

5 *months.*—*Faradic stimulation.* Front of forearm: Good response observed in flexor carpi radialis and in flexor sublimis and flexor profundus digitorum. No response in intrinsic muscles of hand supplied by median nerve. Extension: Good response in triceps and supinator longus only.

10½ *months.*—Unfortunately the baboon died from acute pneumonia before any further examination of the muscular recovery had been made. The microscopical examination of the anastomoses in the upper arm and of the posterior interosseous and median nerves in the upper part of the forearm show that considerable nerve regeneration had taken place.

Microscopical Appearances.—*Fig.* 378, A shows the median-musculospiral anastomosis 10½ months after operation. The distal end of the proximal segment of the median nerve is bulbous. The regenerated fibres in the musculospiral nerve below the anastomosis are finer and take the stain less deeply than the fibres of the median above the anastomosis.

Fig. 378, B shows the musculospiral-median anastomosis. The distal end of the proximal segment of the musculospiral nerve is bulbous. The fibres of the regenerated median nerve below the anastomosis take the stain less deeply below the anastomosis than the fibres of the musculospiral nerve above the anastomosis.

Nerve-fibres pass through both anastomoses, thus explaining the functional results observed.

Fig. 379, A shows regeneration of neurilemma sheaths in the median nerve below the bend of the elbow, while *Fig.* 379, B shows regenerated axis cylinders in the posterior interosseous nerve below the bend of the elbow.

Experiment 12.—**Crosswise anastomoses of the internal and external popliteal nerves.**

RHESUS MONKEY.—The external and internal popliteal nerves were divided. The proximal segment of the internal popliteal nerve was united to the distal segment of the external popliteal nerve, and the proximal segment of the external popliteal nerve was united to the distal segment of the internal popliteal nerve (*Figs.* 380, 381). A thick piece of muscle was placed between the anastomoses. Two sutures of Van Horn arterial silk was used to fix each anastomosis.

2 *months.*—Wasting of leg muscles. No reaction to weak faradic current. Response to strong faradic current: Tibialis anticus, extensor communis digitorum, and peronei; all flexor muscles of leg, and the abductor pollicis of the foot. Response to galvanic current: Slow contraction of all muscles.

4 *months.*—Animal ill, hence was killed by ether anæsthesia. Before death the nerves were exposed and stimulated with the faradic current.

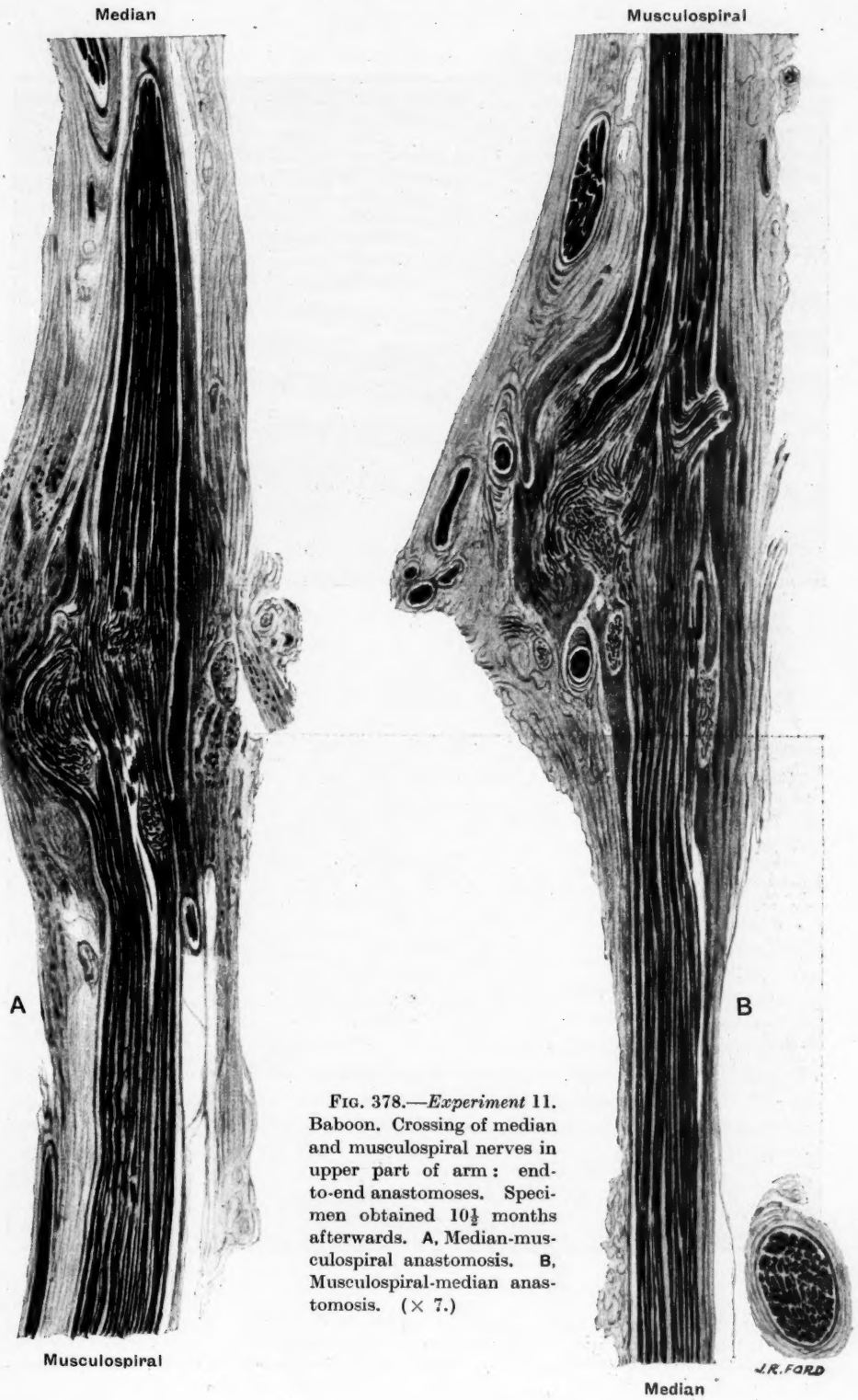
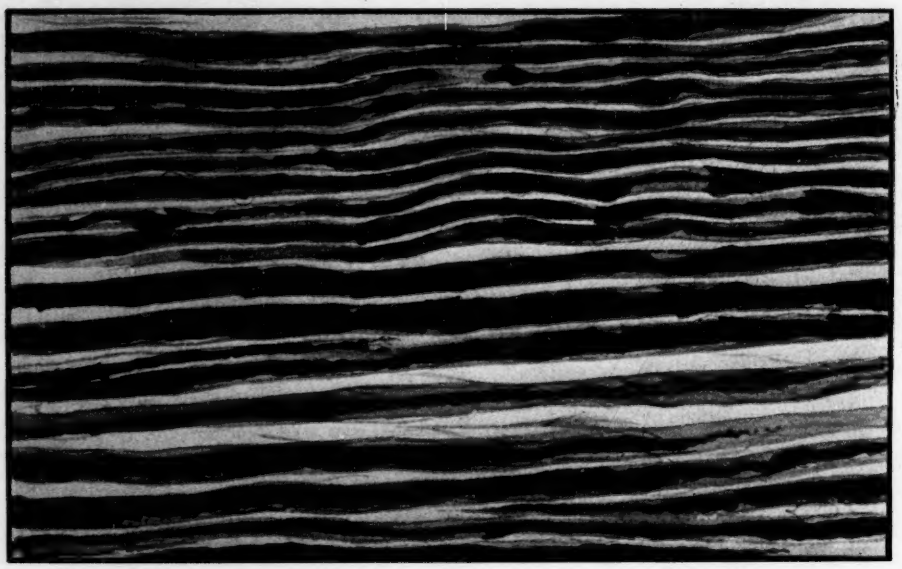
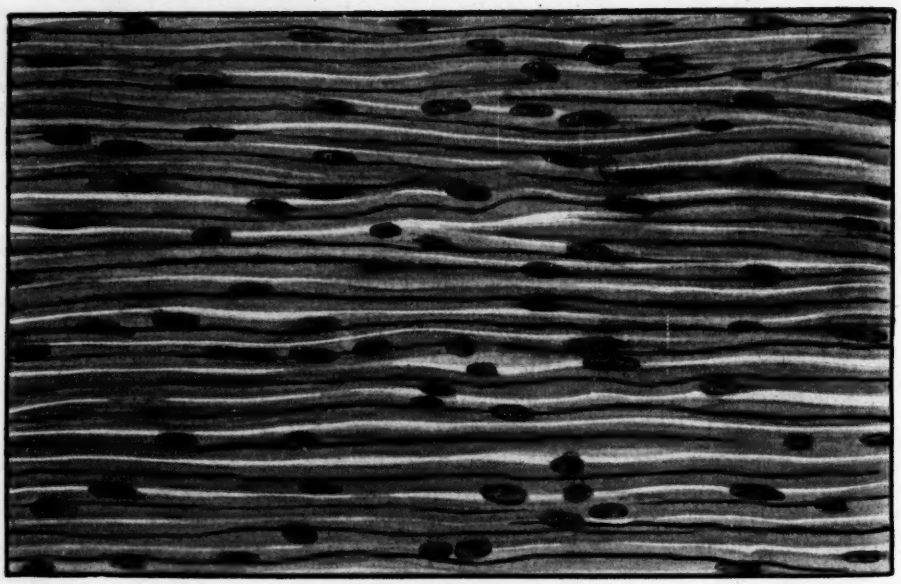


FIG. 378.—*Experiment* 11. Baboon. Crossing of median and musculospiral nerves in upper part of arm: end-to-end anastomoses. Specimen obtained 10½ months afterwards. A, Median-musculospiral anastomosis. B, Musculospiral-median anastomosis. (× 7.)



A



B

FIG. 379.—From the same experiment as *Figs.* 377 and 378. Portions of the median nerve (A) and the posterior interosseous nerve (B) removed below the bend of the elbow. The sections show regeneration of both nerves. A was stained with Weigert, B with Stroebe. (× 450.)

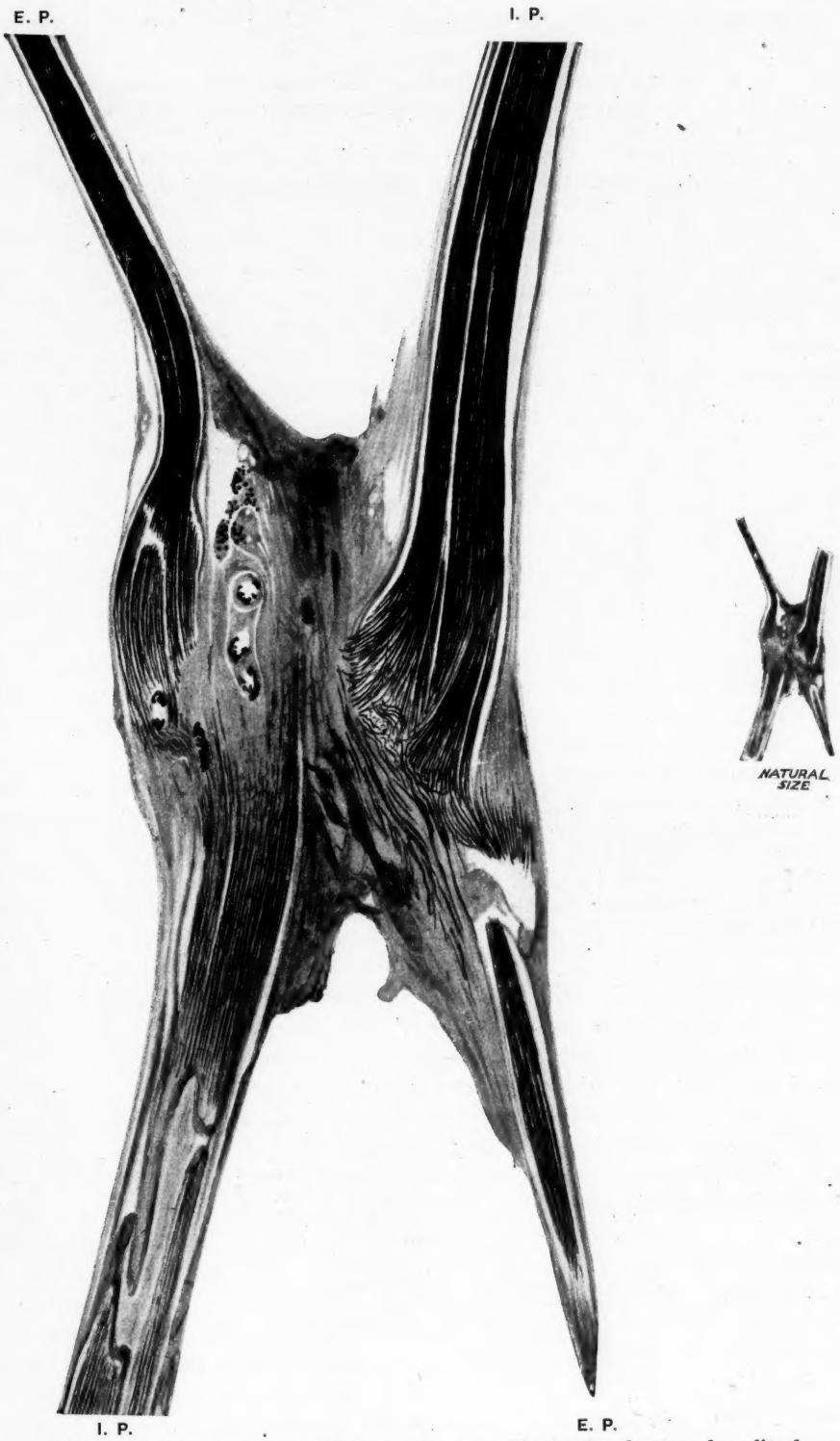


FIG. 380.—*Experiment* 12. Monkey. Crossing of internal and external popliteal nerves in the popliteal space: end-to-end anastomoses. Specimen obtained 4 months afterwards. (× 7.)

Results of Stimulation (*Fig.* 382) :—

A, Flexion of toes and ankle.

B, Strong extension of ankle. The toes were curled up owing to shortening of the flexor tendons and muscles, but the extensors of the toes contracted.

The monkey died before stimulation of the appropriate areas of the opposite Rolandic cortex for extension and flexion of the ankle could be carried out.

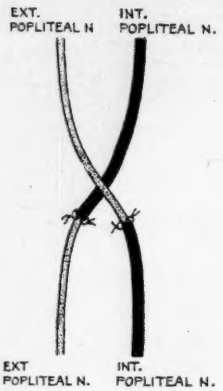


FIG. 381.—*Experiment* 12. Diagram showing crosswise anastomoses operation of the internal and external popliteal nerves.

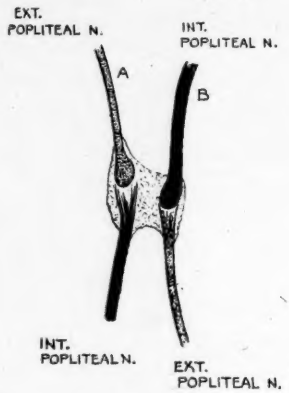


FIG. 382.—*Experiment* 12. Diagram showing the points on the popliteal nerves at which faradic stimulation was applied, and also the parts removed by dissection after death. 4 months.

Microscopical Appearances.—Fig. 380 reveals the fact that union has taken place between the external popliteal and internal popliteal nerves and between the internal popliteal and external popliteal nerves respectively at the sites of the two anastomoses. The distal ends of the proximal segments of the external and internal popliteal nerves are both bulbous. In the regenerated segments of both popliteal nerves distal to the anastomoses the fibres are finer, more numerous, and less deeply stained than the fibres in the nerves proximal to the anastomoses.

ILEUS FOLLOWING FRACTURED RIBS.

By F. G. RALPHS, Dukinfield.

Uncomplicated fracture of the ribs is a surgical commonplace; its association with grave abdominal symptoms is of sufficient rarity to make it worthy of record.

The patient was a store-keeper, age 60, who was admitted to hospital with the following history. Six days previously he had slipped on a frosty pavement, falling heavily on his right side. He was conveyed home, and strapping was applied to the chest by his doctor. In the ensuing days, the bowels failed to respond to the usual aperients, though prior to the injury he had never been subject to constipation and was accustomed to a daily stool. The day before admission it was noticed that his abdomen was distended, and he was removed to hospital.

On removing the strapping, crepitus and surgical emphysema were noted in the right axilla. A skiagram revealed ununited fractures of the sixth and seventh ribs midway between their angles and sternal ends. There was general distention of the abdomen, with a tympanitic note on percussion. The distention was most marked centrally. Neither tenderness nor rigidity was present, and rectal examination proved negative. The tongue was thickly furred and dry, temperature normal, pulse 90. An enema gave a small fæcal result with the passage of flatus, but the distention persisted despite the administration of pituitrin and eserine. By the following day, his condition had obviously deteriorated. The distention had increased, the pulse was quick and weak, and another enema failed to relieve the absolute constipation, now of twenty-four hours' duration.

The condition had apparently developed into one of acute obstruction, and operation was decided upon. His transit to the theatre was interrupted by a copious vomit of dark-brown intestinal contents, of fæcal odour. Under novocain anæsthesia, enterostomy was quickly performed through a median incision, two inches in length, just above the umbilicus. The base of the first distended loop of small intestine which presented was sutured to the parietal peritoneum, and the gut opened. There was an instant escape of flatus and gush of liquid fæces, soiling the wound thoroughly before a catheter could be tied in the bowel for drainage.

During the next twelve hours, eighteen ounces of fluid fæces drained away, and as the abdominal distention persisted, another enema was given, with the expulsion of much flatus. On the second day after operation, the patient had four normal motions per rectum, there being no drainage through the enterostomy tube. On the third day, eighteen ounces escaped through the tube, the bowels did not move naturally, but much flatus was passed after a turpentine enema. By the evening of the third day the distention

had completely disappeared. On the fourth day the patient plucked the tube from his wound without untoward result, the fæcal fistula draining freely into the dressings. About this time he exhibited restlessness and mental confusion. From the fifth day onwards the bowels were moved naturally, and the amount of drainage from the fistula steadily diminished. Five weeks after operation it was completely closed, and the wound soundly healed.

During convalescence a systematic examination of the gastro-intestinal tract was carried out by opaque meal and enema. No lesion was found to account for the symptoms of obstruction, nor was any delay noted in the passage of the meal through the intestines. Hence the condition which supervened has been regarded as one of paralytic ileus. Since discharge from hospital sixteen months ago the man has remained steadily at work and enjoyed good health without recourse to aperients.

OBSERVATIONS.—A search of the literature has revealed a single reference to a like condition following upon rib fracture. Some years ago, Mr. J. E. Adams,[1] in a paper, "Paralytic Ileus as a Sequel of Fractured Ribs", described two cases similar to the above, in one of which he employed the same method of treatment by ileostomy. In both the fracture was close to the angles of the ribs, and was therefore in the region of the sympathetic cord and ganglia lying on the necks of the ribs. Arguing from this relation, Adams inferred that "some displacement of the proximal fragments occurred sufficient to irritate the sympathetic chain", and, *pari passu*, the splanchnic nerves arising from the lower half of the gangliated cord in the thorax. Now Starling[2] has shown that stimulation of these nerves produces inhibition of peristalsis in the small intestine. To splanchnic irritation, then, was ascribed the train of abdominal symptoms which occurred in the two previously reported cases. It is an ingenious theory, but difficult of comprehension from the anatomical standpoint. The head of the rib is so securely anchored by short and strong ligaments as to prohibit, one must think, the range of movements assumed for it by the hypothesis above. As stress has been laid by Adams on the site of the fracture, it is of interest to note that in the present case it was situated at some distance from the angle, viz., in the axilla.

Again, cases of multiple fracture of ribs are not uncommonly found in the accident wards of hospitals. Yet how infrequent is the occurrence of the abdominal condition outlined in this case! Perhaps some other explanation might be attempted. Maybe the rib fracture and the intestinal paralysis are alike the results of a common cause, i.e., the accident, and not dependent the one upon the other. Now it is known that, with the splanchnics intact, inhibition of the movements of the small intestine follows on stimulation of the sensory nerves of the abdomen. An every-day demonstration occurs to us in the temporary inhibition of peristalsis which follows an abdominal section. A slight distention of the intestine with gas results, and usually begins to pass off in twelve to twenty-four hours after operation. Should it persist, the conditions would be favourable for the onset of paralytic ileus. It may be argued in the cases under consideration that a similar inhibition of peristalsis is produced through irritation of the abdominal sensory nerves by the injury.

To explain why this paralytic condition should endure, and terminate in ileus, some other factor seems necessary. Perhaps it is a vascular one, not peculiar to the territory of the intestines, but involving the systemic circulation as a whole. A review of the clinical features of these three cases shows that in two of them a note was made of the altered mental condition of the patient. Thus, in the first case, reported by Adams, it is described as "abnormal", and "mental depression was still a marked feature of the man's condition". In the present case, similar mental changes have already been described. This simultaneous exhibition of cerebral and abdominal symptoms leads one to suggest that, following upon the rib fracture, traumatic lipæmia and fat embolism may have been a causal factor in the production of the sequelæ described. Of the presence of this in the case under consideration no evidence can be adduced, from the regrettable omission to make systematic examination of the blood and urine during the man's stay in hospital. Thus it is merely speculative, and presented, with all reserve, as an explanation of the perplexing symptoms which may rarely follow simple fracture of the ribs.

REFERENCES.

[1] ADAMS, J. E., "Paralytic Ileus as a Sequel of Fractured Ribs," *Ann. of Surg.*, 1910, li, 102.
[2] STARLING, *Recent Advances in the Physiology of Digestion*, 1905.

PLASTIC OPERATION FOR COVERING A GUILLOTINE AMPUTATION STUMP.

By W. B. GABRIEL, London.

The methods at present recognized for treating a guillotine amputation stump prior to healing are either: (1) Glue or strapping extension on soft parts, which usually results in a puckered and adherent terminal scar; or (2) Re-amputation if the bone has failed to be covered by the previous method.

The covering of a guillotine stump by a pedicled graft is here suggested as a better method, in that it avoids the necessity of re-amputation and gives a much firmer stump than can possibly be obtained otherwise without sacrificing a further length of bone.

It is an established principle (Huggins[1]) that in a forearm stump at least three inches and sometimes four inches of ulna are necessary to enable a forearm bucket to be fitted. In the case now to be described with the aid of the accompanying photographs, the employment of a pedicled graft enabled a useful length of a forearm stump to be saved, and provided a firm covering of healthy skin and fascia over the end of the stump. The case was one in which it was particularly important that as much bone as possible should be conserved, seeing that both hands had been lost. Reference to *Fig.* 383 shows that it would have been impossible in the ordinary way to cut adequate flaps and still preserve the necessary length of bone below the elbow to enable an appliance to be fitted.

The patient, a male, age 21, was admitted to the Middlesex Hospital on July 2, 1924, as an emergency, having been involved in a machinery accident. Both hands had been crushed in a rolling machine, and had been held for some minutes before the machinery could be stopped and his release effected. On admission to the ward he presented a moderate degree of shock, being pale and sweating, pulse 64. The wounds had been dressed temporarily in the Casualty Department. Under general anæsthesia three hours later the condition of affairs was found to be as follows:—

Right Arm.—A complete cuff of skin had been torn down from half way up the forearm. The hand was severely crushed: multiple fractures were present; the thumb was almost torn off, and was hanging by the extensor tendons. Skin was avulsed from both aspects of the hand, and the soft parts were heavily infected with dirty material.

Left Arm.—The left hand was quite cold up to the wrist, and white in colour. Skin and subcutaneous tissues were lifted off the palm and part way up the forearm. Soft parts were extruded from two large lacerated wounds, and the hand felt like a bag of bones. The whole of the left forearm was bluish-red in colour.

OPERATION.—After very careful consideration it was decided that both hands were too severely injured to be conserved, and amputation was done through the lower third of both forearms. Short antero-posterior flaps were cut and loosely sutured over the ends of the stumps. Flavine dressings were applied, and antitetanus serum, 1500 units, given subcutaneously.

SUBSEQUENT COURSE.—The patient's general condition remained good, and the right forearm stump healed without trouble. On the left side a discoloration appeared, and finally became established dry gangrene of the distal half of the stump (*Fig.* 383).

On July 16 re-amputation was performed. The knife was carried circularly round the limb at the line of demarcation, without any attempt to fashion flaps. Wet flavine dressings were applied. No further extension of gangrene occurred, and the end of the stump gradually became covered over with healthy granulation tissue.

FIG. 383.—Photograph taken on July 16, 1924, showing gangrene of forearm stump prior to circular amputation.

PLASTIC OPERATIONS.—

1. On Aug. 18 a strip of skin and subcutaneous tissues measuring 6 in. by 2½ in. was lifted from the left side of the abdominal wall, and was resutured in position with its ends intact.

It was decided to perform the 'delayed flap' method in view of its effective result in limiting the blood-supply of the graft to its two extremities, as a preliminary to raising the graft from a single pedicle. The risk of gangrene, which might have occurred if such a long graft had been raised in one stage with its blood-supply dependent entirely upon vessels entering at one end, was thus avoided. At the same time it was a less cumbersome method than that of suturing the graft in the form of a tube, which always results in considerable shrinkage of the graft when the time comes for it to be unrolled.

2. On Aug. 29 the graft was raised again from the abdominal wall and freed at its outer end. The amputation stump was prepared for its reception by making a circular incision at the junction of skin and granulations, and obtaining a clear-cut edge of healthy skin to which the graft was to be sutured. The radius was shortened by half an inch. The pedicled graft was then swung across and sutured down over about three-quarters of the circumference of the stump. The arm was immobilized to the side in a

light plaster case. The bridging part of the graft was sutured to form a tube (*Fig.* 384).

3. Twelve days later, on Sept. 10, under local anæsthesia the graft was severed from the abdominal wall, and after being suitably trimmed it was

FIG. 384.—Circular amputation had been performed at the line of demarcation. The illustration shows the pedicled graft attached to the stump: Sept. 3, 1924.

sutured snugly down so as to cover the remainder of the raw surface entirely. For two days previously the attached end of the graft had been constricted for several hours a day by a rubber tube to encourage its new vascular

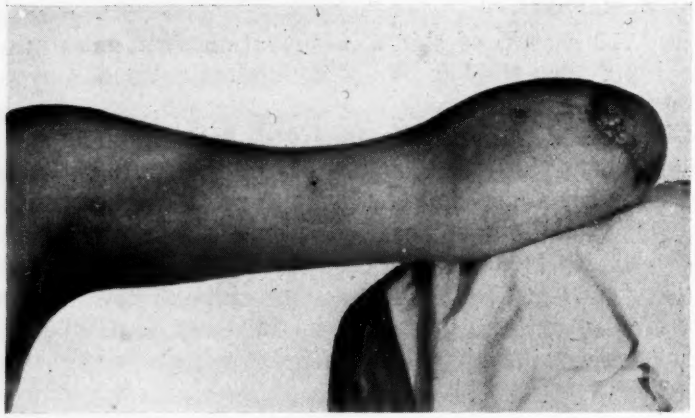


FIG. 385.—Lateral view of the stump: Nov. 7, 1924.

connection, and after its separation no anxiety was experienced in regard to its vitality. *Fig.* 385 shows a lateral view of the stump on Nov. 7.

The patient was discharged on Oct. 3, 1924.

RESULT.—The result has been excellent. The end of the left forearm

has a smooth firm covering: the bones are not adherent, and in fact they have a better covering on the grafted side than on the opposite one. X rays show no sign of infection of the ends of the radius or ulna. There is a full degree of flexion and extension at the elbow, and a moderate amount of pronation and supination. There is a length of $5\frac{1}{4}$ in. of ulna, which is an

FIG. 386.—Photograph of the patient fitted with his artificial arms, showing range of flexion at the left elbow: April, 1925.

ample length for an appliance. *Fig.* 386 shows the patient with the artificial limbs which have been made by Messrs. Grossmith.

I am indebted to Miss Clephan for the series of photographs. My chief, Mr. A. E. Webb-Johnson, was kind enough to allow me to treat the patient throughout his stay in hospital. I am greatly indebted to him for his help and encouragement, and for his permission to record the case.

REFERENCE.

[1] HUGGINS, G. M., *Amputation Stumps*, 1918, Oxford University Press.

SHORT NOTES OF RARE OR OBSCURE CASES.

AN UNUSUAL COMPLICATION IN OPERATING ON LARGE TUMOURS OF THE NECK.

LIEUT.-COLONEL H. HALLILAY, I.M.S., CALCUTTA.

THE following case appears to be sufficiently uncommon to be worth recording.

S. K. B., age 20, Bengali, consulted me in June, 1925, about a large tumour on the right side of the neck which he said had been there since his early childhood. The tumour was a large lobulated mass occupying the whole of the space between the clavicle and the lower jaw on the right side of the neck. There was a striking under-development of the jaw on that side, and a condition of wry-neck towards the left. The right carotid could be traced from near the middle line along the inner aspect of the tumour to where it was lost behind the lower jaw. The larynx was displaced to the left of the middle line. The patient told me that he had been informed that any attempt to remove the tumour surgically would probably be fatal.

OPERATION, June 20, 1925.—Novocain and adrenalin. Transverse incision. It was necessary to tie off a large vein at the lower pole of the tumour, and one blade of a Mayo-Ochsner forceps was slipped under the vein preparatory to dividing it. As the blades were closed, the patient's voice died away into a husky whisper, and his pulse commenced to race. It being perfectly evident that in some way the fibres of the recurrent laryngeal had been caught up in the forceps, it was hastily disengaged, and the vein carefully dissected away from the capsule of the tumour. It was then seen that a nerve, suspected to be and subsequently identified as the vagus, was lying under and concealed by the vein. The nerve was stretched over the anterior surface of the tumour and was imbedded in the capsule, and it was necessary to dissect it out for the whole of its extent before the tumour could be released.

When the tumour was finally enucleated, the nerve was seen to be a much elongated vagus nerve, which, owing to the fashion in which it had been stretched by the growth, was about twice the normal length.

The remarkable feature of the case was the way in which the growth had separated the constituents of the carotid sheath, the right vagus being stretched over the front of the tumour, while the right carotid and jugular were displaced to the left of the middle line. The natural question which arises is, Where did the growth originate?

The tumour was examined by the Pathologist to the Medical College Hospital, Calcutta, Capt. Shanks, I.M.S., and was found to be a lipoma. It

weighed 40 oz. The patient made an excellent recovery, but had some degree of hoarseness and a rapid pulse for some weeks after the operation. The tumour had been diagnosed as a goitre, but the position of the carotid on the left of the tumour put this diagnosis out of court.

In conclusion, the incident affords a further testimony, if any were needed, of the virtues of a local anæsthetic in the removal of these formidable tumours of the neck. Had the operation been done under a general anæsthetic the vagus would inevitably have been divided—a misadventure which at the best would have led to the complete destruction of the patient's powers of phonation, and at the worst might have caused his death. Even the former would have been an irreparable disaster, for the patient intends to enter the Bar.

I have not encountered any mention of this complication in my reading, and would be much interested to learn if any one else has had a similar experience.

SUPPURATING THYROGLOSSAL CYST.

By W. E. M. WARDILL, Newcastle-upon-Tyne.

The following short note of a specimen in the Durham University College of Medicine Museum is offered as illustrating a cause of dyspnœa and dysphagia which was directly concerned with the death of the patient.

A female infant, age 3 months, was admitted to hospital in a state of collapse, with a history of difficulty in swallowing and breathing over a period of five weeks. The child was grossly emaciated, and there was marked indrawing of the intercostal spaces during each inspiration. A rounded, smooth, and elastic mass was palpable in the middle line of the neck immediately below the mandible.

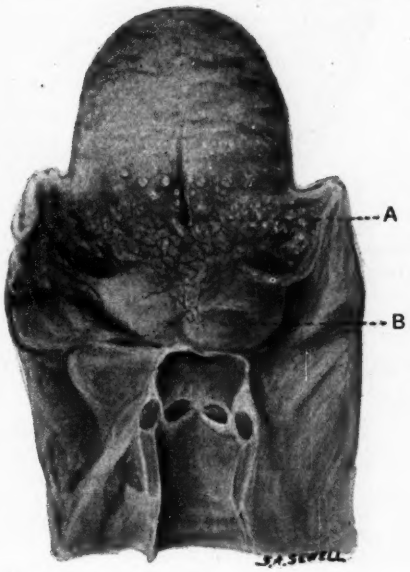


Fig. 387.—Thyroglossal cyst.
A, Foramen cæcum; B, Cyst.

At the autopsy, the specimen shown in *Fig.* 387 was recovered. It consists of a thin-walled cyst, a little larger than a hazel-nut, which projects from the pharyngeal surface of the tongue and extends downwards as far as the base of the epiglottis. The median glosso-epiglottic ligament is stretched over the cyst, and is indicated by a slight longitudinal depression; both valleculæ are obliterated. The cyst is filled with greenish pus, which, in the recent state, could be squeezed through the patent foramen cæcum. The inflammatory process does not extend to the surrounding parts; hence there is no œdema of the glottis. In front the cyst can be seen to form a small swelling limited below by the upper border of the hyoid bone. No abnormality was found in the lower part of the thyroglossal tract.

MUCOUS CYST OF TONGUE.

By ERIC I. LLOYD, London.

The tongue here illustrated was removed post mortem from a girl of four months who was said to have suffered from impeded breathing for over eight weeks. Tracheotomy was performed, but the child died two days after admission to the Hospital for Sick Children, Great Ormond Street.

The illustration (*Fig.* 388) was drawn from the untouched specimen, and shows very well its general appearance. There is a cyst nearly half an inch in diameter in the mid-line of the dorsal aspect of the tongue half an inch behind the foramen cæcum. The cyst impinges against the front of the epiglottis, and by pushing it backwards partly closes the rima glottidis. The tumour is covered by normal mucous membrane, and the glosso-epiglottic fold runs along its superior surface.

Fig. 388.—Showing size of cyst and position at base of tongue. (*Natural size.*)

Fig. 389 shows the mounted museum specimen after hemisection of the tongue and removal of the roof of the cyst. The cyst was found to be thin-walled, and contained a semi-fluid substance which was not examined as the preparation had been long in a preservative solution. The inner surface of the cyst wall was smooth, and no channel or duct leading from it could be discovered. Serial sections were cut with the object of finding the remains of the thyroglossal duct, but no trace of this was seen. The lining of the cyst was formed by a single layer of cubical cells, and external to this were alveoli and tubules of mucous glands. The thyroid and thymus glands were normal.

Fig. 390 is drawn from a section, and shows the microscopic appearances.

Fig. 389.—Hemisection of tongue, showing cyst with its roof removed. (*No.* 1162.2, *R.C.S. Museum.*)

The pathology of this rare congenital abnormality is obscure, but there are three possibilities :—

1. A dermoid cyst arising from an infolding of epiblast in the mid-line during development of the tongue is in some ways similar, but most of the recorded examples have been in the floor of the mouth, and such cysts usually have lining walls of thick epidermis with an appreciable amount of fibrous tissue; they may also contain hair and other epithelial products.

2. A cyst developed in connection with the thyroglossal duct or its remnants may occur anywhere between the foramen cæcum and the isthmus of the thyroid gland; such a cyst is lined by ciliated columnar cells, and is most commonly found in the neck. The duct is normally replaced by a fibrous cord, but is lined, when it remains patent, by squamous epithelium for a few millimetres at its proximal (buccal) end, and distally by ciliated columnar epithelium. Probably in the tongue (1) and (2) are respectively buccal and pharyngeal variations of the same process occurring in different parts of the thyroglossal duct.

3. A mucous cyst may occur beneath the mucous membrane of the posterior third of the tongue where small racemose glands are normally found.

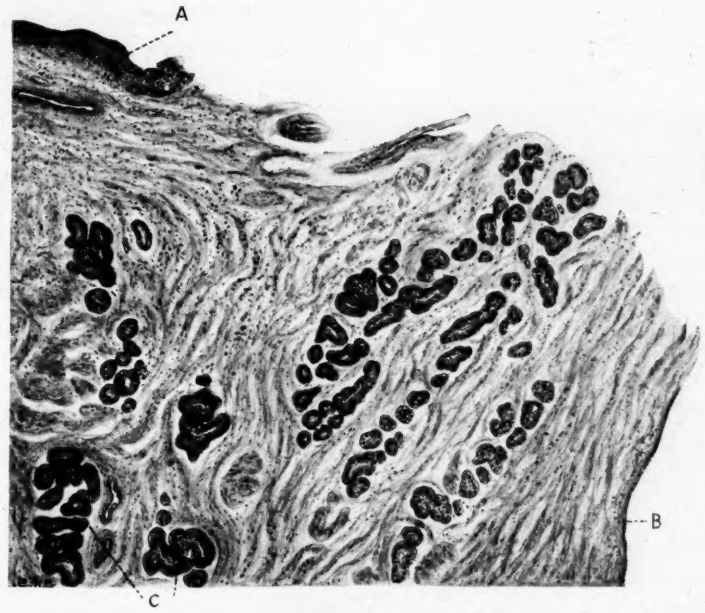


FIG. 390.—Microscopic section through tongue and wall of cyst. A, Mucous membrane of tongue; B, Cyst wall; C, Mucous glands. (× 50.)

Such cysts are presumably due to blocking and subsequent distention of the ducts or alveoli; they are thin-walled, and seldom larger than a pea.

There seems no reason for associating this particular cyst with the thyroglossal duct, and from its position and its naked-eye and microscopic appearances, it must be classed as a mucous cyst.

The Museum of the Royal College of Surgeons of England possessed no example of this condition until Mr. George E. Waugh presented this specimen (1162.2). I am indebted to him for permission to describe what is both a clinical and pathological curiosity, and to Professor Sir Arthur Keith for his help and interest.

RHABDOMYOMA OF THE TONGUE.

By GEOFFREY KEYNES, London.

Tumours composed of muscle-fibres are uncommon in any part of the body other than the uterus, and I am unable to discover a record of any case of rhabdomyoma of the tongue similar to the one here described.

The patient was a traveller, age 26. For about six to nine months he had noticed a small white spot on the dorsum of his tongue. This had occasioned a good deal of irritation and discomfort, which was increased by smoking and had been made worse by the application of silver nitrate to the spot. He was first seen at St. Bartholomew's Hospital on Dec. 17, 1924, when he was found to have a small, hard, rounded lump, 0·5 cm. in diameter, situated in the mid-line of the tongue half-way between the tip and the foramen cæcum. The lump was tender to pressure, but there was no appreciable change in the overlying epithelium, which was only slightly elevated above the surface, and there was no induration of surrounding tissues. A diagnosis of 'fibroma of the medium raphé of the tongue' was made, and the lump was excised under a local anæsthetic, together with a small wedge-shaped portion of the tissues around it.

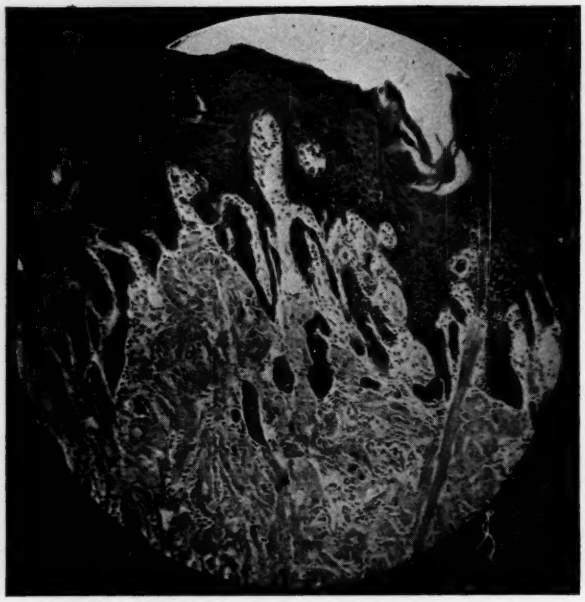


Fig. 391.—Low-power view (× 45) of part of the tumour with the overlying epithelium of the tongue. A bundle of normal muscle cells can be seen among the tumour cells on the right-hand side of the field.

No recurrence of the growth has since taken place, and nine months after the operation the tongue was found to be perfectly normal.

The cut surface of the tissue removed showed a rounded white area, apparently continuous with the overlying epithelium, and infiltrating the surrounding muscles. The microscopic appearances are seen in the accompanying figures. *Fig.* 391 shows a low-power view of about half the tumour. The epithelium over it is quite unaffected, although it is in immediate contact with the tumour cells; these form a rounded mass of large, irregularly disposed cells, staining less intensely with eosin than the normal muscle cells, a few of which are interspersed among the tumour cells without being in any way altered. There is no trace of a capsule at the edge of the tumour, nor is there any sign of an inflammatory

reaction. In *Fig.* 392 is seen a higher magnification of a group of tumour cells. These are large and elongated, with a very granular cytoplasm.

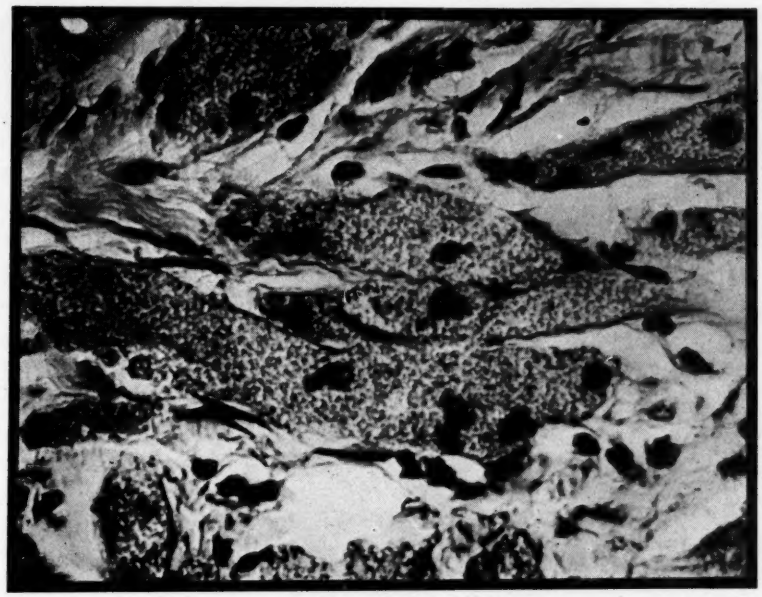


FIG. 392.—High-power view (×560) of a group of tumour cells with associated endothelial and connective-tissue cells.

FIG. 393.—Oil-immersion view (×1040) of a tumour cell lying between two unaltered striated muscle cells.

Fig. 393 shows a still higher magnification of a tumour cell lying between two normal striated muscle cells. Their sizes and appearances may thus be

easily compared. No striation could be demonstrated in the tumour cells, even in sections stained with phosphotungstic acid; but a survey of the rather scanty literature of rhabdomyoma, such as is given in Ewing's *Pathology of Tumours*, shows that the cells are often found in an embryonal form without striation. In *Fig.* 394 are seen, side by side, very high magnifications of oblique sections through a striated muscle-fibre and through a tumour cell. The similarity is striking, though the organization of the tumour cell seems to stop just short of striation.

FIG. 394.—Oil-immersion view (× 1200) of the cytoplasm of a tumour cell (right-hand side) and of an oblique section through a neighbouring striated muscle cell (left-hand side).

In spite of the absence of striation there can be little doubt of the nature of the tumour. If it is subjected merely to a process of elimination, it can only be identified as a rhabdomyoma. Sir Bernard Spilsbury has kindly examined the sections for me with the greatest care, and has confirmed this opinion. The tumour, though not encapsulated, seems to be innocent in character, so that no treatment beyond the conservative operation already performed is likely to be needed in the present case.

The characters of the growth were scarcely well enough defined for a clinical identification of so rare a tumour to be made on a future occasion. The diagnosis must rest rather on the histological appearances.

The microphotographs illustrating this article have been made by Miss M. Vaughan, Dunn Laboratories, St. Bartholomew's Hospital.

CYSTIC PNEUMATOSIS OF THE SMALL INTESTINE.

BY S. H. PUGH, TRAVANCORE, S. INDIA.

THE following case of cystic pneumatosis of the small intestine will be of interest, especially in view of the article by Cyril A. R. Nitch in the April, 1924, number of the JOURNAL.

Male Indian, age 39. Patient said he had vague symptoms of dyspepsia for the past fifteen years. There were, however, 'short intervals' when his digestion was good. For the last three years he had definite pain three to five hours after food, which was worse at night. He first noticed distention of the abdomen twenty months before admission. This became progressively

worse during the first ten months. During the second ten months the condition remained about the same. He vomited once or twice a month during the last three years, but never more frequently. The vomit rarely contained food, but usually consisted of 'bile or mucus'. He had long been losing weight.

ADMISSION.—March 29, 1924. The patient was emaciated, his weight being 78 lb. During the nine days he was kept under observation, his temperature was normal, and pulse varied from 68 to 84. There was slight distention of the abdomen. Constipation was not marked. There was no vomiting, but the appetite was poor.

OPERATION.—April 8. An exploratory laparotomy was performed through a mid-line incision above the umbilicus. Immediately the peritoneum had been incised, I was astonished by an audible escape of gas, and for an instant thought the intestine must have been opened. This was obviously not so, and the gas was odourless. That there had been no previous perforation of the stomach or intestine was also indicated by the absence of any sign of acute inflammation. The parietal peritoneum, and serous covering of the stomach, upper part of jejunum, and omentum were perfectly normal. There were no adhesions anywhere, and no free fluid in the abdominal cavity. No lesion was found in the stomach or duodenum. The small intestine, however, presented a remarkable appearance. From a point about three or four feet from the duodenojejunal flexure, on the surface of several sections of the bowel from two or three inches up to about three feet in length there were numerous blebs of all sizes up to about one-third of an inch. The blebs obviously contained gas, for when they were pricked they immediately collapsed, and apparently contained no fluid. Many of the blebs were exceedingly thin and transparent like soap bubbles. It is difficult to believe that these delicate blebs could remain long without breaking and liberating their contained gas. This would indicate that the gas was being formed at a considerable rate. Several of the affected sections of the bowel were greatly and irregularly thickened. On handling they were more rigid and heavier than normal. It was surprising that the action of the bowel had not been more interfered with. The increased thickness of the bowel suggested that there had been a long-continued but very mild irritation. There was no sign of congestion or indication that there had ever been any acute inflammation. The last three inches of the ileum and the whole of the large intestine appeared to be unaffected. So much of the bowel was involved that resection was impossible, and the abdomen was closed. The patient recovered from the operation, and his condition, when he left the hospital three weeks later, was much the same as when he was admitted.

This case is apparently the first of the kind reported in India.

Nitch draws attention to the association of gastric or duodenal ulcer in 50 per cent of the recorded cases. Duodenal ulcer is exceedingly common in Travancore. During 1924 in the Neyyoor Hospital the operation of posterior gastro-enterostomy was performed for that condition in 129 cases. While, therefore, one is familiar clinically with the condition and with the appearances at operation, in this case neither the symptoms nor the findings at the operation suggested gastric or duodenal ulcer. It seems unlikely, therefore, that the gas under pressure, such as that derived from aerophagy, etc., was

forced through an abrasion in the stomach or duodenum, especially in view of the fact that the ileum was more affected than the jejunum. It is, however, certain that the disease caused some, if not very much, obstruction in the small intestine. Yet it is difficult to believe that the obstructed gases were forced through any lesion in the intestinal mucosa. That a very large volume of gas was formed, and that its rate of formation was rapid, was indicated by the fact that the gas was being produced as fast as it could be absorbed even under considerable pressure by the serous surfaces in the abdomen. Would it be possible for so much foul air to escape from the bowel into the tissues without bringing acute inflammatory consequences in its train, and for the gas itself to become quite odourless, when it had reached the abdominal cavity?

Then the marked local thickening of the intestine, and the rather long history of the case, indicate that the irritating influence remained confined to the particular affected parts for a considerable period of time. It seems hardly likely that the same channel or channels for the escaping gas would remain patent for any great length of time, and so open into the same tissues.

On the mechanical theory it is evident that each bleb must have its own separate ultimate channel from which it receives its gas. That would necessitate many hundreds of channels, derived perhaps by branching from the main channel, sufficiently small to pass the gas slowly enough to give time for the formation of the blebs and to allow them to become of large size before they burst. It seems unlikely that the mechanical conditions could be so exactly reproduced in so many and widely separated parts of the bowel as to bring about the widespread similar thickenings of the bowel wall, and a great number of blebs of just the same appearance. This similarity of effect seems to suggest that the affected tissues were almost equally permeated with the cause of the condition. This is more suggestive of a chemical or bacterial agent than of a mechanical explanation.

On the other hand, if the mechanical explanation is not the true one, it is clear that the agent which produces the gas so rapidly must be a very active one. If the cause is bacterial, it is difficult to reconcile such great bacterial activity with the complete absence of all signs of acute inflammation. Moreover, if the presence of 15 to 20 per cent oxygen in the gas is confirmed in other cases than those of Krummacker and Urban quoted by Nitch, that would involve the difficulty that the microbe must be an oxygen-former. These two serious difficulties in the bacterial theory would seem to make the mechanical theory the least improbable explanation so far offered, in spite of the difficulties associated with that theory.

Since writing the above I have heard from the patient's friends, twelve months after the operation, that he improved a good deal soon after leaving the hospital. Practically all the symptoms disappeared, and he was able to enjoy ordinary food until three and a half months ago, when the old symptoms began to recur "with vomiting and eructations of gas [this may suggest aerophagy, which is not uncommon in Travancore], the vomiting becoming gradually worse, though there was some abatement for a short time, and he has now become so ill that he cannot leave his bed without help". His friends promise to bring him again to the hospital if he improves sufficiently, when further observation should be of interest.

A CASE OF IMPERFORATE ANUS WITH MEGALOCOLON AND TERMINAL PERITONITIS.

By R. WATSON JONES, Liverpool.

There are many features of interest in the case of M. L., female, age 3½, who came under treatment at the Royal Infirmary in the early months of this year. The mother's complaint was that the child had no anus, and suffered from an incontinent discharge of fæces from the vagina. A history of constipation was elicited, with occasional attacks of diarrhœa, at which intervals the motion was inclined to be foul and offensive. Otherwise there were no symptoms, and the mother's worry was mainly a cosmetic one.

Examination.—An examination revealed the presence of an anal dimple but with no communication with the rectum. Vaginal inspection showed an opening in the posterior wall, at the junction of the middle and lower thirds, through which fæces were discharged. Some stress must be laid on the fact that the opening was not in the vulva, but well up in the vaginal wall above the hymen, which was present. The vagina was not septate and appeared otherwise normally developed.

Operation.—This was undertaken by Mr. Thelwall-Thomas, and as a first stage the rectum was brought down by perineal dissection to the position of the normal anus, opened, and stitched there. The wound healed rapidly, and the child was discharged. Two months later she was re-admitted, and an attempt was made to close the vaginal fistula from which fæces were still discharged in spite of the fact that the new anus was functioning. Convalescence progressed satisfactorily for three weeks after the second operation, when the patient suddenly became ill and vomited persistently. The abdomen was rigid and distended, and showed reversed respiratory rhythm. Laparotomy confirmed the diagnosis of general peritonitis and showed a markedly dilated and hypertrophied colon. On the third day the patient died.

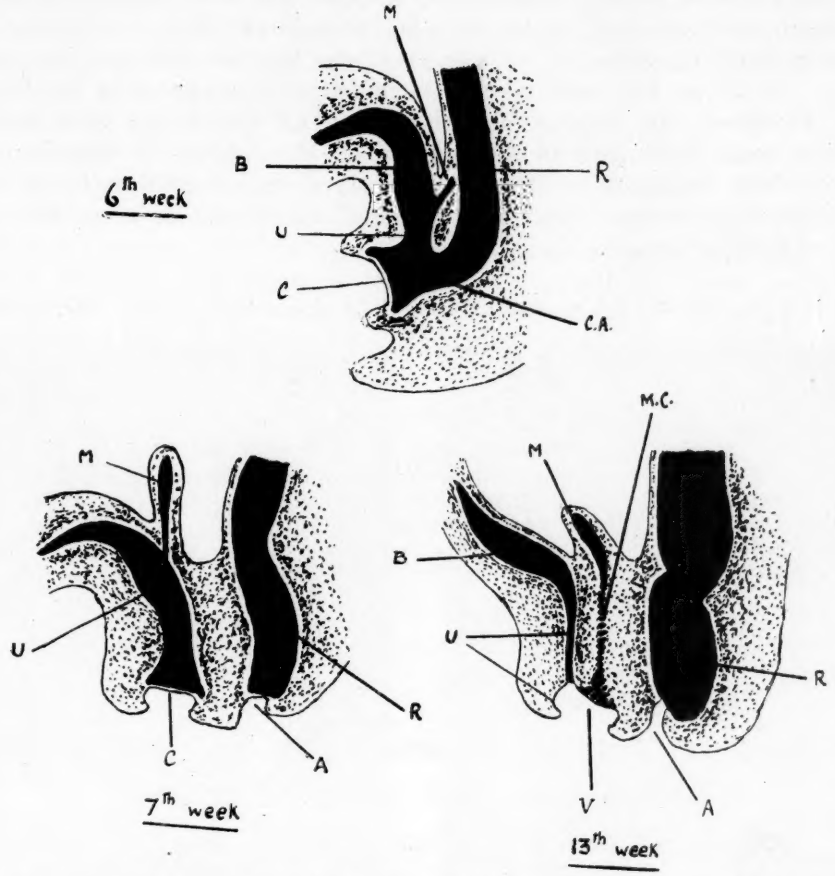
The case is of interest in the sudden onset of the final stage without any obvious direct cause. The patient had lived three and a half years without developing any signs of chronic obstruction with the sole exception of constipation and occasional diarrhœa. She was a fairly healthy and well-nourished baby; there had been no vomiting, no abdominal pain, no passage of mucus or blood, no abdominal distention, and there was no visible peristalsis. Presumably the vaginal opening was adequate, and certainly after operation the new anus was as efficient as, if not more efficient than, the previous orifice—its efficiency being ensured by the daily passage of a large-sized rectal tube, and the daily administration of an enema. Actually, however, there was a widely dilated and hypertrophied colon, containing large scybalous masses, though the dilatation was limited to the iliac and pelvic portions of the large intestine, the rest of the alimentary tract being normal. Although the probabilities are in favour of the condition being in reality that of chronic intestinal obstruction, the resemblance to congenital megalocolon (Hirschsprung's

disease) is marked. The definite hypertrophy shows that the condition was not of recent development, and the fact that the enlargement was strictly limited to the sigmoid colon and rectum is suggestive. It seems unlikely that the final events were ushered in as a result of the obstruction becoming more complete; indeed, the reverse seems to be the case; moreover, the clinical and post-mortem findings were those of a primary serofibrinous peritonitis, not of an acute intestinal obstruction. One is therefore led to the conclusion that two operations and hospital life had reduced the resistance of the child to the abnormal bacterial decomposition that had always been going on, so that the mucous membrane and walls of the colon had been invaded and a general peritonitis produced.

Another interesting anomaly in this case was revealed at autopsy, illustrating the truth of the statement that congenital abnormalities are frequently multiple. A prolonged search failed to discover the presence of a gall-bladder, the common bile-duct leading directly from the hepatic ducts. Section of the liver proved that there was no gall-bladder hidden by a complete pons hepatis, and there was no sign of the organ in the gastrohepatic omentum or elsewhere.

The case, moreover, is instructive from the embryological standpoint. It is a not uncommon combination to find with an imperforate anus the rectum opening into the vulva immediately posterior to the hymen. This corresponds with the most frequently found condition in males, where the rectal orifice is in the verumontanum of the prostatic urethra. It is rare, however, for the communication to be high up in the posterior vaginal wall. Keith[1] has described such cases, and states that in his experience they are associated with a septate vagina—presumably the presence of the rectum having interfered with the normal process of fusion of the Müllerian cords. In this case the opening was at the junction of the middle and lower thirds of the vagina. Such a condition cannot be accounted for by the generally accepted explanation of its development—namely, that first given by Wood Jones.[2] The theory is made clear by the diagrams below taken from Wood Jones's thesis (*Figs.* 395–397).

In the seventh week of development, the rectum migrates backwards, its cloacal orifice disappears, and the urogenital sinus is left with the Müllerian ducts opening into it. The vagina, however, is developed from a completely new structure, a down-growth of solid columns of mesodermal cells from the lower ends of the Müllerian ducts. Wood Jones believes that the whole of the vagina is formed in this way, by a canalization of these Müllerian cords. This will explain the condition where the rectum opens into the vulval cleft, for the vulva is certainly developed from the urogenital sinus. But how comes the rectum to communicate with the vagina if this be formed entirely from the Müllerian cords which are a later development, and if the urogenital sinus produces nothing more than the base of the bladder, the urethra, and vulva? The only satisfactory explanation is that in some cases at least, as suggested by Blair Bell,[3] the lower third of the vagina is developed from the posterior part of the urogenital sinus, cut off from the anterior part by the vesicovaginal septum. The Müllerian cords perforate the summit of this portion and form the upper two-thirds of the vagina (*Fig.* 398).



FIGS. 395, 396, 397.—Diagrams to show development of vagina according to Wood Jones. B, Bladder; U, Urogenital sinus; M, Müllerian ducts (uterus); R, Rectum; C.A., Cloacal anus; C, Cloacal membrane; A, Anus; M.C., Müllerian cords; V, Vulva.

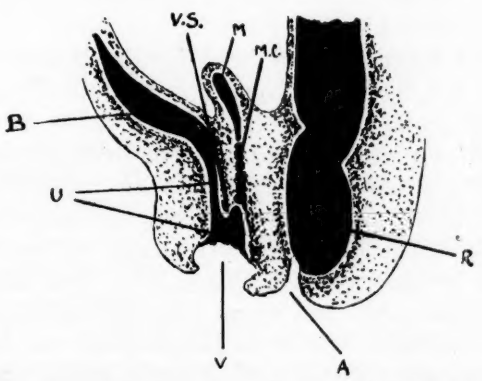


FIG. 398.—Diagram to show development of lower part of vagina from urogenital sinus—which permits of a vaginal opening of the rectum, being explained as a persistent cloacal anus. B, Bladder; U, Urogenital sinus; M, Uterus; M.C., Müllerian cords; V.S., Vesico-vaginal septum; V, Vulva; A, Anus; R, Rectum.

Such must be the explanation of the development in the case described, even if it is not true of all cases. The fact that the level at which the rectum may open into the vagina varies only illustrates the fact that varying proportions of the urogenital sinus may be included in the vagina in its development. Moreover the hymen cannot consist of the fringe left after the Müllerian cords have broken through on to the surface[4, 5]—that is, at the junction of the vagina with the urogenital sinus—but must develop in association with the breaking down of the cloacal membrane or urogenital plate, which originally closes the sinus below.

It is a pleasure to acknowledge my indebtedness to my chief, Mr. Thelwall-Thomas, for permission to investigate the case at autopsy and to publish the findings.

REFERENCES.

[1] KEITH, "Malformations of the Hind End of the Body", *Brit. Med. Jour.*, 1908.
[2] WOOD JONES, "The Nature of the Malformations of the Rectum and Urogenital Passages", *Brit. Med. Jour.*, 1904, Dec. 17.
[3] BLAIR BELL, *The Principles of Gynæcology.*
[4] BERRY HART, *Edin. Med. Jour.*, 1911, vi, 577.
[5] KEITH, *Human Embryology and Morphology*, 4th ed., 377.

PULSATING GOITRE ACCOMPANIED BY RECURRENT DISLOCATION OF THE EYEBALLS.

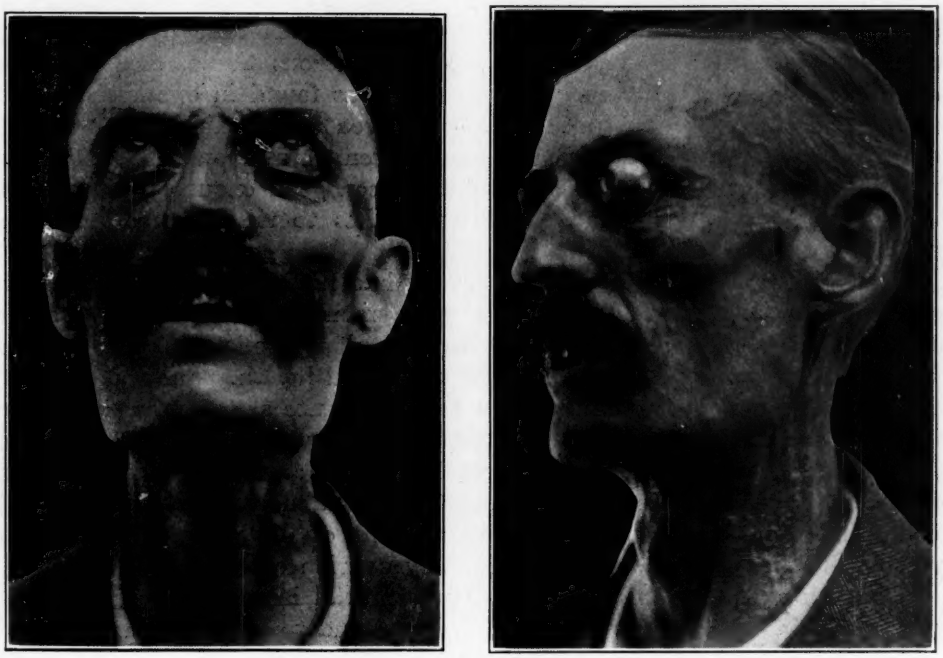
BY HAROLD BURROWS, PORTSMOUTH.

DISLOCATION of the eyeball, though it has been known to happen in little Pekinese dogs, and perhaps in some other animals, seems to have been a rare occurrence in mankind. Gradual forward displacement of the globe by an orbital tumour is, of course, a recognized condition; but the case about to be described is of another kind.

The patient, now aged 45, had been invalided out of the Police Force in 1918 after sixteen years' service, owing to a gradually increasing weakness which prevented him from getting through his day's round of a large rural district which included a military camp. He first consulted me in August, 1923, on account of repeated dislocation of the eyeballs, stating that any effort such as sneezing or yawning, or merely looking downwards suddenly to the ground, would cause his eyeballs to come out of their sockets unless he put up his hand to keep them in place. For the same purpose he used to throw his head backward, and these two precautionary movements had become habitual with him. The man himself was emaciated, and suffered from pulsating goitre, over which a loud bruit could be heard. This bruit was audible over the front of the chest as low down as the fifth costal cartilage in the nipple line on each side, and in both right and left interscapular regions; it was conducted also on to the cranium. Exophthalmos,

though pronounced, was not extreme (*Fig.* 399, *a*). Tachycardia was present in a moderate degree, the pulse-rate when he was at rest being usually at about 92 or 96. His weight was 8 st. 12 lb. He suffered from the usual nervousness and tremors of Graves' disease.

The most interesting feature of the case was the ease with which the eyeballs could be dislocated by digital pressure on the upper and lower lids (*Fig.* 399, *b*). When a dislocation had been produced in this way, the patient at once effected reduction by placing the pulp of his forefinger on the cornea and pressing the eyeball back into its proper position. It was manifest that the dislocation of the eyeballs caused him some momentary



a *b*

FIG. 399.—*a*, Shows moderate exophthalmos and wide ocular clefts; *b*, Shows the left eyeball dislocated through the lids.

uneasiness and apprehension, and he lost no time in effecting reduction in the manner just described. His eyesight was unimpaired.

Partial thyroidectomy was performed on Nov. 15, 1923, the eyelids being stitched together in order to prevent the occurrence of a post-operative dislocation. These stitches, which caused a good deal of discomfort, were removed on the following day.

When seen in April, 1925, the patient's weight was 11 st. 6 lb., tachycardia had disappeared, and he said he could do anything. No dislocation of the eyeballs had taken place since the operation, although some degree of exophthalmos was still to be observed.

THYROID GLAND TISSUE IN DERMOID CYST OF OVARY.

By ERNEST H. SHAW, London.

The following case of dermoid ovarian cyst is recorded on account of its unusual contents. As far as the writer can remember, it is the first specimen in which he has discovered thyroid tissue in a dermoid. The microscopic examination of teratomata is always interesting, and the varied picture seen in many of them affords the morbid histologist great pleasure. It seems so odd and weird to see bits of the various organs and tissues of the body jumbled together in one section—skin with its various appendages of sebaceous glands and hairs, mucous membrane with nice columnar-celled glands, striped and unstriped muscle, fat and fibrous tissue, etc. Even an incomplete ring of choroid with its pigments was found in one specimen.

A small portion of the specimen was received for examination and report from Dr. H. F. Ealand, who has kindly given permission for publication. Mr. Eric Sheaf, who removed the cyst, has been good enough to supply the clinical and other details, and the writer wishes to thank these gentlemen for their kindness.

The patient, Mrs. D., age 71, nullipara, had noticed a gradual and painless enlargement of the abdomen for some months. This had caused no inconvenience except some fullness. No difficulty of micturition or defæcation. There had been no loss of weight or general health. She was found to be a spare healthy old woman, with a tumour in the abdomen, rising out of the pelvis and extending above the umbilicus. It was smooth, and movable laterally as far as its size permitted, tensely elastic in consistence, and dull on percussion. No dullness in the flanks or other abnormality was made out. No vaginal examination was made on account of difficulty caused by the hymen being intact. Diagnosis: 'Probably ovarian cyst'.

Operation.—July 31, 1925. A left paramedial incision exposed a bluish cystic swelling. On delivery its pedicle was found to be connected with the left broad ligament. The pedicle was tied, and the tumour removed in the usual way. The uterus and right ovary were normal except for a few very small fibroids in the uterus.

Macroscopical Examination of the cyst after removal showed it to be composed of several loculi, some containing thin fluid, one near the base with thicker wall containing the white thick oily material and hair characteristic of dermoids. On one side of the wall was a solid mass composed partly of cartilage, from its hardness ossified in places, and some soft tissue not unlike that of thyroid gland.

Microscopic Examination.—Layer of skin, thrown into folds, with a large number of sebaceous glands. Two hair roots are seen cut obliquely across. A few sweat glands are present in the underlying fat. A small patch of ill-formed bone is seen at one end of the section, and below this, and separated from it by a band of fibrous tissue, there is a large patch of thyroid tissue. Most of the thyroid vesicles are large and filled with colloid material. In some places the vesicles are quite small. (*Figs.* 400 and 401.)

FIG. 400.—Illustrates the skin elements, with the large number of sebaceous glands which are so common in dermoids.

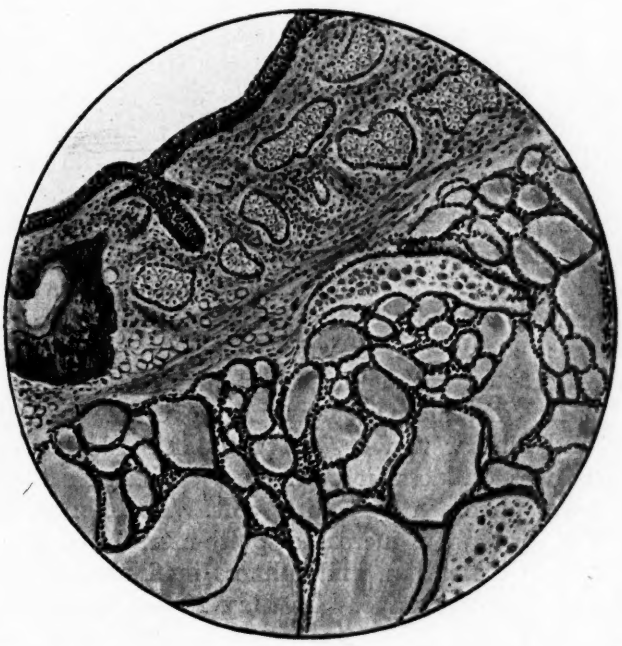


FIG. 401.—Also shows skin structures, with a dark patch of ill-formed bone on the left. Below this is a mass of typical thyroid vesicles. Two collections of brown-coloured cells indicate old blood extravasation. This is interesting in view of the fact that hæmorrhage is so common in thyroid adenomata.

TRAUMATIC RUPTURE OF THE HEPATIC DUCT.

By S. A. WALLACE and A. SPIRO, London.

The rare occurrence of this condition, and the difficulty of diagnosis and deciding on the proper course of treatment, has led us to report this case.

The patient, W. G., age 47, motor driver, was crushed between two motor-cars on the morning of May 12, 1925. He was brought to the Acton Hospital in a condition of profound shock. Temperature 96·8°, pulse 36, respiration 24. He could give a rough account of the accident, and complained of pain in the right hypochondriac region. No pain below the umbilicus or in the left side of the abdomen. On examination, the right upper rectus muscle was rigid, and he was tender over the gall-bladder area, but the liver dullness was normal. On account of the liver area receiving the main impact of the crush, and the pain and tenderness being localized here, a diagnosis of rupture of the liver was made.

His shock was treated, and in a few hours he improved immensely. Opinion was divided as to the course of action, the fact of his having had a serious injury being outweighed by the great improvement in his general condition; his temperature was now 98°, pulse 50, and respiration 20, and it was decided to leave him alone. During the first night one noticed he was slightly jaundiced, and in the morning this was quite marked; there was blood and bile in the urine. He was free from severe pain, however, and his general condition was good; temperature 99°, pulse 74. On May 14 he was feeling very comfortable, but the jaundice was deeper, and there was bile in the urine and fæces. On May 16 the jaundice was very deep; the bowels were acting normally, but the stools were clay-coloured for the first time. The patient's general condition was good; in fact, he wanted to get up. On palpation, his rigidity and tenderness were the same.

He remained quite comfortable until May 21, when he complained of more pain in the gall-bladder area; he was not resting so well, and his general condition was much worse. His pulse was over 100 for the first time; tongue dry and coated, eyes sunken, breathing jerky, with troublesome hiccough at times. He was intensely jaundiced; urine loaded with bile, but no blood present; stools clay-coloured. He appeared more drowsy, but could get no real rest on account of the pain and hiccough. The abdomen showed some general distention; very tender over the epigastrium and right hypochondriac and lumbar regions. Liver dullness down about 1½ in., and in addition there was dullness in both flanks, which shifted on moving the patient. The lower abdomen was fairly soft. The clinical signs which had developed on this day were increased pulse-rate, hiccough, jerky type of breathing, abdominal distention, and fullness in flanks, all showing that the condition had now become grave. On May 22 the patient was still weaker, and the above signs and symptoms were a little more marked. Blood-pressure: systolic, 155; diastolic, 90. He was seen by Dr. R. A. Young, C.B.E., who suggested a laparotomy in the hope of finding something which might be remedied.

OPERATION, May 23.—By Mr. C. H. Shorney Webb. Mid-line incision and also a transverse incision to the right. About 3 pints of fluid containing blood and bile in peritoneal cavity. A few minutes after the abdomen was opened the patient suddenly stopped breathing. Artificial respiration and massage of the heart were unavailing, and death followed.

POST-MORTEM EXAMINATION.—All the tissues and viscera were deeply bile-stained. The liver was much enlarged; the gall-bladder was partly full and intact,

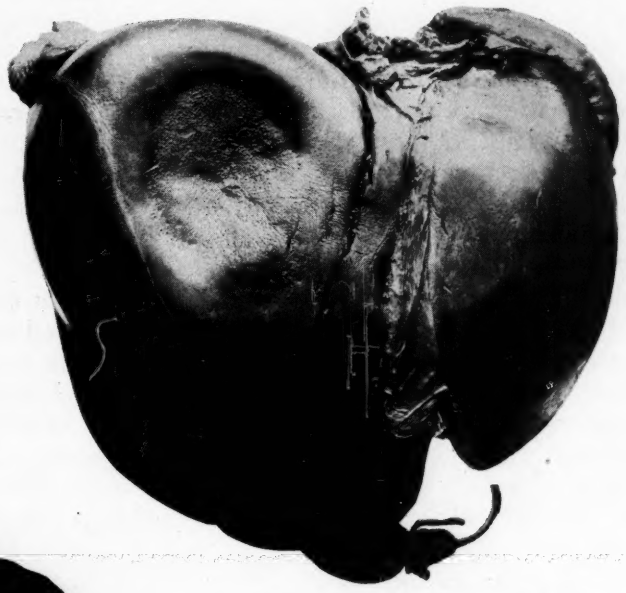


FIG. 402.—Anterior surface of the liver, showing ruptures.

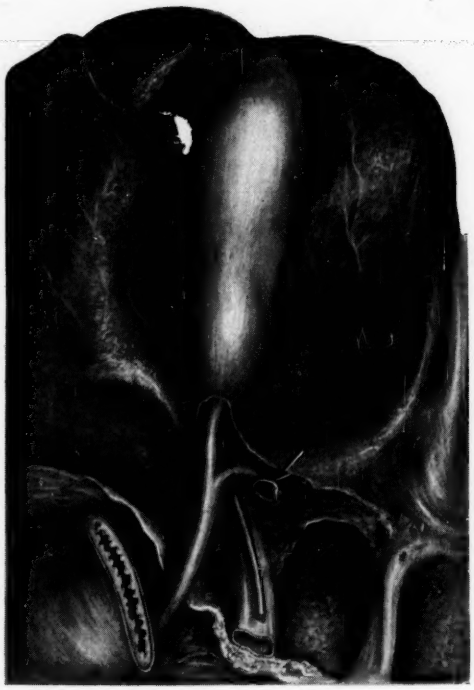


FIG. 403.—Diagram showing inferior surface of the liver. The arrow indicates the rupture in the hepatic duct. The hepatic artery can be seen lying on the portal vein.

and the stomach and intestines were unruptured. On the anterior surface of the liver was a rupture about 8 inches long, just to the right of the attachment of the falciform ligament, and immediately to the right of this were two smaller ruptures each about 1½ inches (*Fig.* 402). These ruptures varied in depth from ½ to 1 inch, and were in the process of healing, being filled in by partly organized blood-clot which was adherent to the walls of the ruptures; this prevented much leakage of blood or bile during the latter part of the patient's illness, but the leakage from the ruptures must have been considerable immediately after the accident. There was a large tear in the anterior layer of the lesser omentum near the attachment to the transverse fissure, and by careful dissection here a tear in the hepatic duct which would admit the blunt end of an ordinary lead pencil was found (*Fig.* 403). This rupture of the hepatic

duct was everted, and there was some organization of the surrounding blood-clot. It was through this that the bile was escaping into the peritoneal cavity, where it was absorbed into the blood-stream.

Comment.—There are very few cases of rupture of the hepatic ducts reported in the literature; since 1900 we can find only 15 recorded cases of traumatic rupture. Garrett reports a very interesting one in 1900. A farmer of 21, crushed between timber, was severely shocked, but after this passed off was comparatively well for ten days, when he became jaundiced and distended. He was aspirated twice, and in all 16 quarts were withdrawn. Laparotomy was performed on the twentieth day, and a rupture of the proximal part of the common bile-duct found. Drainage was provided and complete recovery followed. He also quotes a case of Spencer's, in which a child run over by a cab became jaundiced on the third day, and at operation 1½ pints of bile-stained fluid were withdrawn and the abdomen was drained. The abdomen was re-opened on the eighteenth day for a collection of fluid within the peritoneum. Death occurred on the thirty-third day, and post mortem the gall-bladder was found torn off. Porter reports a case which died forty-eight days after operation, with large sub-diaphragmatic abscess, and the common bile-duct was found completely severed. Hildebrandt mentions two cases in 1906. One, a child of 5, received a crushing injury; laparotomy showed rupture of the hepatic duct, and with drainage recovery was complete in four weeks. Waugh recorded an interesting case in 1915 in the BRITISH JOURNAL OF SURGERY. A boy of 6 was run over by the wheel of a car. There were no symptoms for three days, when he became jaundiced and distended, and bile was found in the urine and clay-coloured stools were present. At laparotomy 1½ pints of bile and blood were found in the peritoneum, and a tear of the anterior layer of the gastrohepatic omentum, the portal vein being plainly seen in this, but the common bile-duct was not seen. Drainage was provided, but seven days later the patient was jaundiced and distended, and the abdomen was re-opened and 2 pints of bile-stained fluid found in the lesser sac. This was repeated a month after the first operation, and recovery was complete in the end.

In studying these cases one is struck with the fact that they all have several important points in common. Following the severe collapse resulting from the injury there is a variable period of comfort and freedom from symptoms. Jaundice is the first sign to appear—in our case before the end of 24 hours; in Garrett's case it did not appear till the tenth day; the average is about three days (Desrosier). The reason for the period of quiescence is that normal bile is only slightly infectious and causes little peritonitis, except in cases of gall-stones, where a fatal peritonitis may be quickly set up (Keen). Later on more serious symptoms arise, such as increase of pain and tenderness, rigidity, distention, and vomiting. The absorption of bile into the blood-stream by way of the peritoneum has made the patient toxic and has damaged especially the heart muscle—probably by direct action of bile salts on the cardiac muscle—and the patient is now a very poor operative risk.

The recorded cases show that some recover when operated on some time after the accident (Garrett's case was operated on twenty days after), but the prognosis is shown to be fairly good if operation is done shortly after the injury, when the only signs present are the jaundice and some tenderness and rigidity in the region of the liver. Meyer states that 4·75 per cent of ruptured livers go into jaundice; but even if at operation only a rupture of the liver is found, the drainage of the peritoneal cavity is a beneficial thing, and at the same time the rupture could be attended to. When rupture of the bile-duct is found, repair, if possible, should be attempted; but if this is impossible, or the tear cannot be found, drainage is sufficient, as the rupture heals in time.

We are indebted to Mr. C. H. Shorney Webb for permission to publish this case, and for his kind advice and assistance.

HERNIA THROUGH THE FORAMEN OF WINSLOW: SECONDARY RUPTURE THROUGH THE GASTRO-HEPATIC OMENTUM.

By E. ROCK CARLING, London, and A. TENNYSON SMITH, Orpington.

One of us (A. T. S.) was called to see a man, A. M., age 27, at midnight on Aug. 2, 1925, when he was complaining of severe pain in the upper abdomen. The day before he felt perfectly well; for supper he had a surfeit of dripping; on the morning of the 2nd he was sick, and vomited several times during the day. At 8 p.m. he was seized suddenly with severe pain in the abdomen; it persisted without relief.

When seen at midnight he was sitting up in bed, unable to lie down, and had recurrent paroxysms of colic. He had not the facies of the acute abdomen. Pulse 68, temperature 97°. He referred the pain to the upper zone of the abdomen, and said that it was worse when he tried to take a deep breath. His bowels had been open that afternoon. The abdomen was slightly distended in the upper half; there was tenderness in the epigastric region, but to no marked extent, and the muscles above the umbilicus were slightly rigid; they relaxed, however, when one engaged him in conversation till the next paroxysm of 'bursting pain' came on. There was no tenderness in the lower abdomen. Liver dullness normal. He could not sit forward for examination of his back, but rolled over in a three-quarter prone position. There was a tympanic note in the right posterior dorsal region extending up almost to the angle of the scapula, and his breath sounds were more distant than on the left side. He seemed to find relief from the semi-prone position; the pain left him, but returned when he resumed his former posture.

He was sick again during the night. He was admitted to the Cray Valley Hospital on the morning of the 3rd; by this time, though the local

tenderness was unaltered, the whole of the abdominal muscles were on guard and rather masked the distention noted the night before.

We saw the patient together at 10 o'clock on the morning of Aug. 3. He was half-sitting in bed, rocking himself to and fro in unbearable pain; it seemed located mainly in the upper zone of the abdomen, which was extremely rigid. There was but slight distention and no visible swelling; there was hyper-resonance over the liver area. It was doubtful if there was even the narrowest band of hepatic dullness just below the chest resonance, and in view of the fact that there was no general distention, this sign was interpreted as due to free gas, and a provisional diagnosis of leaking gastric ulcer was advanced.

On opening the abdomen, free fluid, slightly blood-stained but not turbid, was found; the narrow stomach lay noticeably low, and was reddened and distended; the small intestine immediately came into view above the lesser curvature. The loop came from behind through a gap in the gastro-hepatic omentum. Attention was at once directed to the foramen of Winslow, whence a much distended, and a collapsed, coil were found issuing. By combined pressure through the lesser sac and traction from the foramen the herniated gut was extracted. Though there were petechial hæmorrhages on the surface of the coil and on the mesentery, the bowel was viable, and was returned to the general abdominal cavity over the front of the transverse colon. The rent in the lesser omentum, which appeared to be quite recent, was repaired, but œdema around the foramen was such as to make suture very hazardous, whilst the abdominal wall was rigid and straining, and it was not attempted.

Before the abdomen was closed, the parts had resumed a normal arrangement and the only thing noticed about the transverse colon was that the great omentum was slight, scanty, and fat-free. The coil herniated was ileum rather above the mid-point of its extent, and its mesentery seemed to be a little unduly long, with perhaps rather a restricted attachment area on the posterior wall; but examination was certainly difficult. The length of bowel in the lesser sac was about 20 inches.

Post-operative recovery was uninterrupted; the man left hospital after a fortnight, and has since returned to his work.

Ullman,[1] in recording a case of his own, has recently given abstracts of twenty-nine others; he includes eight not mentioned in Rendle Short's[2] list, but omits two or three there alluded to; finally, there is McKenzie and Wood's[3] case, the figure from which, with slight modification, would do very well for ours. Amongst all the cases there is only one in which secondary rupture through the lesser omentum occurred, that of Schmilinsky.[4] In Blandin's[5] case the herniated gut re-entered the greater sac through the transverse mesocolon.

REFERENCES.

[1] Ullman, *Surg. Gynecol. and Obst.*, 1924, xxxviii, 225.
[2] Short, A. Rendle, *Brit. Jour. Surg.*, 1925, xii, 456.
[3] McKenzie and Wood, *Ibid.*, 613.
[4] Schmilinsky, *Deut. med. Woch.*, 1919, xlv, 477.
[5] Blandin, 1824, *Traité d'Anatomie topographique*, 467.

ACUTE ASCENDING PARENCHYMATOUS ENTERITIS CAUSING PARALYTIC ILEUS.

By ZACHARY COPE, London.

Any light which can be thrown on the question of paralytic ileus should be welcome, so that the recounting of the following case may prove of interest :—

F. B., a man, age 28, was taken with acute abdominal symptoms on March 18, 1924. When first I saw him on March 21, he was clearly suffering from acute appendicitis. At the Bolingbroke Hospital on that date I removed a very acutely inflamed and gangrenous appendix which lay against the right wall of the pelvis, under cover of a hood of inflamed omentum. The contaminated omentum was removed, together with the appendix, and drainage by rubber tube instituted. All went well for three days, but on March 24 the patient began to vomit copious quantities of fluid, which soon became fæculent. There was no pain, and the pulse remained at 88, but collapse soon supervened ; the cheeks became sunken, the pupils dilated, and as soon as I saw him it was clear that there was no time to be lost before remedying the obstruction. On re-opening the wound the last coil of the ileum was found obstructed and adherent to the right wall of the pelvis. It appeared difficult to clear it, so enterostomy, with short-circuit of the base of the obstructed loop of gut, was carried out. Next morning the patient was much better, and the enterostomy had acted well, but later in the day the flow almost ceased. Vomiting re-started, and the patient again became collapsed. The abdomen was now opened higher up and enterostomy performed in a coil of jejunum. At this operation it was noted that the lower part of the small intestine was enormously thickened with inflammatory œdema, and an attempt was made to open the gut above the inflamed area. There was no evidence of peritonitis. In order to lessen the amount of fluid lost, the suggestion put forward by Wilkie was adopted, and the upper and lower enterostomies were connected by tubing which permitted the fluid escaping from the one opening to re-enter the bowel by the other. For three days the patient's condition improved, but on March 28 the upper tube ceased to drain well, and the patient vomited pints of bile and again looked almost moribund.

The upper wound was then re-opened, and it was found that the thickened œdematous condition had spread farther up the small intestine, beyond the enterostomy. The second enterostomy was therefore closed, and a third one made still higher up at a point where the jejunum was closely in contact with a perfectly healthy-looking transverse colon. It was specially noted that there was no peritonitis, and the contrast between the distended, very thickened jejunum and a contracted, normal-looking colon which was lying adjacent to it was very striking. To make drainage of the small bowel more complete, a portion of the upper jejunum was also anastomosed to the transverse colon. The stomach was also washed out.

At each of these three operations two pints of saline were given intravenously to combat the shock. At no period did the patient complain of

any pain. From that time improvement began, and after a prolonged convalescence, during which the enterostomies were closed, recovery took place. A recent X-ray photograph shows that there is no obstruction, though attacks of colicky pain arise occasionally.

The above case illustrates a condition which I do not think has hitherto been recognized or described. From observation of the gut at the different operations, it was clear that there was an ascending inflammation of the small bowel, starting from the primary site of obstruction, involving the whole thickness of the bowel wall, and gradually ascending almost to the duodeno-jejunal junction. There was no peritonitis, for the large bowel was normal. The absence of pain was presumably due to the paralysis of the nerves in the gut wall by the inflammatory process. The condition in no way resembled those cases of ascending peritonitis which are well known and which have been well described by Handley; it was clearly due to an enteric and not a peritoneal infection. As the state of inflammation extended higher up the gut, the intestinal paralysis reached one enterostomy after the other. The walls of the bowel as observed at the operation were in remarkable contrast to the condition seen in peritonitis. Instead of being thin and almost transparent, they were very thick and œdematous, but not so congested or inflamed in appearance as occurs in peritonitis with ileus.

The corollary might be added that it is sometimes unwise to remove protective omentum in cases of appendicitis if the risk of adhesions is thereby increased.

It is impossible to appraise correctly the influence that the jejunocolic anastomosis had in this case. It was carried out because the writer knew Mr. Handley had achieved success in ileus duplex by a somewhat similar anastomosis.

A drawback which may be attributable to this short-circuit is that the young man now has a portentous appetite, and the bowels are opened rather soon after a meal.

CONCLUSIONS.

1. A condition of acute ascending parenchymatous enteritis may follow mechanical obstruction of the lower end of the small intestine.

2. The inflammation involves all the coats of the bowel and causes paralytic ileus.

3. The condition is unaccompanied by pain, owing presumably to the toxic effect of the products of inflammation on the nerve-endings in the bowel wall.

4. For its cure a high enterostomy is needed.

REVIEWS AND NOTICES OF BOOKS.

Operative Surgery: Covering the Operative Technique involved in the Operations of General and Special Surgery. By WARREN STONE BICKHAM, M.D., F.A.C.S., New York. Vol. VI and Index Volume; pp. 908, with 1224 illustrations. The whole work complete in six volumes, and free index volume. In complete sets only. London and Philadelphia: W. B. Saunders Co., Ltd. Per vol. 50s. net.

THE six volumes of this work on operative surgery are a testimony to the erudition, to the patient industry, and to the sound judgement of the author. Not only are the ordinary, and indeed many of the unusual, operations described, but there are chapters which may be regarded as forming almost special treatises dealing with the operations upon the pelvic viscera of the female; upon the eye; and upon the ear, nose, and throat. We can think of no work published in modern days which so completely covers so extensive a field. There are a thousand pages, on the average, to each volume, and there is one illustration to every page. Such a range of subjects is rarely attempted by a single author; we expect nowadays to find a multitude of writers each dealing with a subject he has made his own. Dr. Bickham's book avoids the discrepancies and the overlapping and the bare patches which every encyclopædia by many authors seems always to display. Yet it cannot be said, in fair criticism, that any part of the vast subject dealt with in the six volumes is inadequately considered.

In a preface that is perhaps overlong, the author's intentions are disclosed. Though he rightly places a high importance upon technique, upon craftsmanship, he recognizes that judgement in the proper selection of a procedure applicable to a particular case outweighs all other considerations. The number of alternative methods described is sometimes a little bewildering, and to the unpractised surgeon this is a disadvantage. No wise guidance is given as to the most apt procedure in such and such conditions, and the earnest student may rise from a careful reading of any chapter knowing exactly in what precise fashion many operations can be done, but undecided as to which one ought in any instance to be selected.

There are omissions here and there—strange omissions sometimes—and there are operations described which should find no place in any modern work. Examples of the former are found in the chapter dealing with thoracic surgery, and of the latter in connection with the surgery of the peripheral nerves.

No detailed description of this work is possible in limited space; but we can give a very cordial welcome to these volumes. They will be of the greatest help to every advanced student, and they may well find a place upon the shelves of the surgeon who needs from time to time to refer, especially for teaching purposes, to the approved methods of other surgeons. The illustrations and letterpress are excellent.

The Early Diagnosis of the Acute Abdomen. By ZACHARY COPE, B.A., M.D., M.S. Lond., F.R.C.S., Senior Surgeon to Out-patients, St. Mary's Hospital; Senior Surgeon to the Bolingbroke Hospital; late Hunterian Professor, and Arris and Gale Lecturer, Royal College of Surgeons. Third edition. Pp. 233 + xiv, illustrated. London: Oxford Medical Publications. 10s. 6d. net.

THE fact that this book has already reached its third edition is sufficient proof of its value as a guide to the early diagnosis of acute abdominal disease.

The section on acute pancreatitis has been entirely rewritten. The symptoms of this disease are divided and described under five headings: (1) Those due to

inflammatory tension in the gland; (2) Those due to swelling of the pancreas; (3) Those due to extravasation of blood; (4) Those due to deranged gland function; and (5) Other symptoms. The author emphasizes the fact that profound shock usually accompanies the pain, and that the pulse is rapid and weak. This immediate rapidity and weakness of the pulse is to our mind one of the most important signs of the disease, and serves as an important factor in the differential diagnosis between this condition and perforation of a gastric or duodenal ulcer, in which we maintain there is no immediate rapidity and weakness of the pulse. Amongst other symptoms mentioned are pain in the loins and cyanosis. In the large majority of cases the pain in the back is intolerable, and is one of the most prominent symptoms in this condition. Cyanosis of the face and extremities, and in many cases of the skin of the abdomen, occurs so frequently that it may almost be taken as a pathognomonic sign.

In the preface to the third edition the author mentions our criticism of his 'stage of primary shock' in perforation of a gastric or duodenal ulcer. It is very necessary to define what is meant by 'clinical shock' in cases of this kind. We believe it is incorrect to describe a patient as suffering from clinical shock in the absence of a rapid pulse and lowered blood-pressure. In the first and second editions of the work this stage was said to last for "an hour or two". In the third edition the author states that "it may last for but a few minutes or persist for an hour or two".

We think that this is an important alteration, as we believe that this stage, if it exists at all, only lasts for a few moments, and in no case have we seen it persist for an hour or two. It is important to emphasize this point, as, if the author's teaching be followed, many will look for the signs of clinical shock—that is, rapid pulse and lowered blood-pressure—in the early stage of perforation, and failing to find them may delay operation at this the most hopeful period.

The author quotes from Moynihan's book on duodenal ulcer thus: "When perforation occurs there is usually a sudden onset of the most intolerable agonizing pain. This pain is hardly exceeded in severity by any that a human being can suffer; the extremity of agony is reached. So profound may the instant impression be that death results. . . . The patient is always prostrate with agony, he looks pale and faint, his face wears a deeply anxious expression, the eyes are wide and watchful, beads of sweat stand out upon his brow, and lines are quickly graven on his cheek". This, he says, could hardly be bettered as a picture of clinical shock, if there be any significance in the word 'shock'. We believe there is some significance in the meaning of the word 'shock', and we believe it to involve, amongst other signs, a rapid pulse and a lowered blood-pressure, neither of which is mentioned in the above quotation. The whole point seems to be a definition of the word 'shock'. True, the patient may look 'shocked' in the social sense of the word, but from a medical point of view—and surely the work is written for the benefit of medical men—the two main signs of clinical shock are absent. We have never yet seen a case presenting the signs of clinical shock as we understand it within a few moments of perforation. We have taken the pulse-rate and the blood-pressure in a large number of these cases, but have failed to find either a rapid pulse or a lowered blood-pressure in the early stages of perforation. One case perforated in the X-ray room. He was in great pain and looked pale and ill, but his pulse-rate and blood-pressure, which were taken immediately, were normal.

We feel that it cannot be too strongly emphasized that patients in the early stage of a perforation do not have a rapid pulse or lowered blood-pressure. The pulse is one of the first things a practitioner observes in an acute abdominal catastrophe, and if he discovers it to be normal and does not realize that it usually is normal in the earliest stage in perforations, he may lose valuable time in opening the abdomen.

Apart from this one point we are in entire agreement with the author's views as set forth in this excellent and helpful little book. It will continue to enjoy the position it has gained, and we can strongly recommend it to all who are interested in this subject. It is undoubtedly the leading book of its kind on this subject to-day.

Abdominal and Pelvic Surgery for Practitioners. By RUTHERFORD MORISON, Hon. M.A. and D.C.L., Hon. LL.D., M.B., F.R.C.S. Ed. and Eng., Consulting Surgeon, Royal Infirmary and Dental Hospital, Newcastle-on-Tyne. Crown 8vo. Pp. 212 + x, with 9 line drawings. 1925. London: Oxford Medical Publishers. 8s. 6d. net.

THIS small book is intended chiefly as a guide to diagnosis, and not as a guide to treatment. The author is impressed with the fact that the diagnosis and treatment of serious disease is now left largely in the hands of specialists, and further that the patient only seeks the advice of such specialist under the guidance of uninformed instinct. He would reinstate the general practitioner into the important position which he held more than thirty years ago, when the family doctor was always the first to be consulted about illness, and made it his business to make a diagnosis and then to advise consultation with a specialist if necessary.

The matter and manner of the book are most excellent. Brevity, clearness, and simplicity mark every page, and illustrative examples are given of all the conditions described.

The Radiological Examination of the Male Urethra. By G. L. S. KOHNSTAM, M.R.C.S., and E. H. P. CAVE, M.B., B.S., etc., late of King's College Hospital. With a Preface by Sir JOHN THOMSON-WALKER, O.B.E., M.B., C.M., F.R.C.S. Pp. 116 + xvi, with 64 illustrations. 1925. London: Baillière, Tindall & Cox. 15s. net.

THE authors of this monograph have applied to the investigation of the male urethra the methods employed by Voelcker and Lichtenberg in the study of the ureter and the pelvis of the kidney. They have, however, abandoned the use of sodium bromide as too irritating, and have finally selected lipiodol, diluted with four times its volume of sterile paraffin. An excellent account is given of the technique of the injection and of the arrangement of the patient and the X-ray tube during the taking of the skiagrams; the latter detail is of importance owing to the difficulty of getting an unobstructed view of the deeper parts of the urethra. A description of the appearances presented by the normal urethra is followed by chapters on deviations from the normal as shown by 'urethrograms' of cases of stricture, of false passages, urethral calculi, of the prostate before and after operation, and of sphincteric inefficiency.

Amongst many interesting observations we would single out as of special interest: (1) Urethrograms showing that, after suprapubic prostatectomy, sphincteric control appears to be taken over by the remains of the prostatic urethra rather than by the compressor urethræ; Sir John Thomson-Walker, in his preface, is of opinion that further observations are necessary before accepting this rather startling innovation. (2) The frequency with which the injection flowed into the vesiculæ seminales. (3) The appearance of relaxation of the bladder neck, called 'funnelling' by the authors, which is found associated with some cases of growth of the bladder and with some spinal diseases; this might prove of real diagnostic importance in cases where cystoscopy is difficult.

There are numerous radiograms and some tracings; the latter are very much easier to understand than the former. The authors are to be congratulated on an excellent piece of pioneer work.

Facial Surgery. By H. P. PICKERILL, C.B.E., M.D., M.S., Surgeon-in-Charge, Facial and Jaw Department, Dunedin Hospital. With an Introduction by Sir W. ARBUTHNOT LANE, Bart., C.B., M.S. 4to. Pp. 162, with many illustrations, and frontispiece in colour. 1924. Edinburgh: E. & S. Livingstone. 21s. net.

THIS volume represents a very laudable effort to concentrate within the confines of a comparatively small book, at a reasonable price, the extensive experience of the author in plastic surgical methods. We hold the opinion that this task is an impossible one, for this branch of surgery has increased so rapidly in its scope and literature since the war, and necessarily requires such profuse illustration by diagrams and case records, that it would seem to us better that an author should confine himself to the treatment of some particular branch of the work in any single volume. A

great many of the cases recorded were treated at the Queen's Hospital, Sidcup, where the author had charge of the New Zealand section. In a rapidly developing and comparatively new branch of surgery it is always difficult to decide to whom the credit of introducing some new procedure belongs: it is difficult to believe that the author was entirely responsible—working as he did in close association with the heads of the other sections of the hospital—for all he lays claim to. There is, however, no doubt that great credit is due to him for having modified and simplified the Esser principle of pressure skin-grafting.

The work is well arranged in three parts: principles, methods, and technique of plastic surgery; military facial surgery; facial surgery in civil practice. The illustrations are very well reproduced. We fail to see the use of that on p. 4, in which "one hundred and forty life-size wax casts of typical facial injuries" are depicted crowded within the dimensions of a single page.

We do not agree with the author in his condemnation of the method of treating gunshot wounds of the mandible by fixation of the fragments in correct relative position to the maxilla, and we do not clearly understand his use of the words in italics, 'accurate approximation'. We feel that it would be better to get non-union in correct position—treating the case later by bone-grafting—than to obtain union in malposition by approximating the fragments. No mention is made of the difficulty of retaining the edentulous short posterior fragment in good position during the process of repair.

We are of opinion that skull defects are much more easily and economically filled by celluloid plates than by grafts from the tibia. The latter invariably become fibrous, and frequently give considerable trouble by becoming adherent to the dura and brain scar. It is well known that a very large number of grafts of this type have called for removal for this reason, and in most of these cases celluloid plates have been substituted with success.

Concerning syphilis of the nose, no mention is made of the need for replacing the loss of mucous membrane—the probable cause of the bulk of the deformity—by skin-grafting before inserting a cartilage graft to restore the bridge line.

A Text-Book of Surgical Pathology. By C. JENNINGS MARSHALL, M.D., M.S., F.R.C.S., Assistant Surgeon to Charing Cross Hospital and to the Victoria Hospital for Children, and ALFRED PINEY, M.D., Ch.B., M.R.C.P., M.R.C.S., Director of the Institute of Pathology, Charing Cross Hospital. Demy 8vo. Pp. 469 + vii, with many illustrations. 1925. London: Edward Arnold & Co. 21s. net.

THIS is a student's text-book of surgical pathology, and will no doubt serve its purpose extremely well. There was need for a new work on this subject, and the collaboration of an active surgeon and working pathologist is calculated to make the book a success. The matter is well arranged, and the information supplied just what the student requires. Many of the illustrations are reproductions of photographs, and show so little that they might well have been left out. This is a pity, as good drawings of pathological specimens are easily obtained.

Malignant Disease of the Testicle: its Pathology, Diagnosis, and Treatment. By HAROLD R. DEW, M.B., B.S. Mel., F.R.C.S. Eng., F.A.C.S., Hon. Surgeon to Outpatients, Melbourne Hospital. Royal 8vo. Pp. 168, with 52 illustrations, including 5 plates in colour. 1925. London: H. K. Lewis & Co. Ltd. 21s. net.

THIS valuable monograph embodies the chief facts put forward in the author's Jacksonian Prize Essay for 1923. It is based on "a review of the literature and a study of forty hitherto unreported cases of this disease". After a brief account of the anatomy and development of the testicle, the author gives an excellent description of the morbid anatomy and histology of testicular new growths. It is pleasant to find that his views tend towards simplification rather than elaboration, for he regards the malignant new growths as virtually of two kinds only, namely, malignant teratoma and spheroidal-celled carcinoma, these occurring with approximately the same frequency. Sarcoma also occurs, but it is so rare as to be almost

negligible. The confusion and uncertainty which have hitherto existed concerning these tumours the author attributes to the many forms of new growth which may arise from the different elements of a teratoma. In considering the question as to the predisposition of a misplaced testicle to malignant disease, the conclusion is reached that this predisposition probably does exist, but has been exaggerated. In his discussion of the differential diagnosis of tumours of the testicle the author insists on the extreme difficulty which may occur in distinguishing a malignant new growth from a gumma. The distinction may be impossible to make, and early operative investigation is urged when the doubt arises.

The results of simple orchidectomy for malignant new growth are stated to be very bad, a certain mortality of over 80 per cent being found. The author therefore advocates the more frequent performance of a radical operation, entailing the removal of the lymphatic channels from the testicle, together with the pre-aortic glands, by a retroperitoneal route. His own experience of this operation at present includes only three cases.

The volume is excellently illustrated with microphotographs and coloured drawings by Mr. H. Mockridge. It should help to shed light in a somewhat dark corner of surgical pathology.

Die Chirurgie. By a number of contributors. Edited by Professor M. KIRSCHNER (Königsberg) and Professor O. NORDMANN (Berlin). Six volumes. Each volume to be published in several parts. To be completed in about two years. Imperial 8vo. With numerous figures in the text and many coloured plates. Berlin and Vienna: Urban and Schwarzenberg. Each part, sewed M.10, bound M.25.

THIS monumental work, of which four parts (forming portions of Vols. I, III, and VI) have already appeared, promises to be a complete account of general and special surgery, written by upwards of sixty German surgeons with the industry and completeness so characteristic of their race. It will form an invaluable store-house of information and be a standard text-book of reference.

Part I, pp. 396 with 193 figures in the text and 2 coloured plates, opens with a general historical introduction by Brunn, taking each section of surgery in turn. It is noticeable that the account of modern surgical development makes little or no reference to any but German workers. Leusden deals with anæsthesia, local and general, laying very great stress on the former.

Boit writes the section on aseptic and antiseptic surgery, including an account of the arrangement of the operating room. Kurtzahn describes the application of the Röntgen rays and radium for purposes of diagnosis and treatment. There are many good figures illustrating typical injuries and diseases, with coloured plates of X-ray lesions. The technique and scope of radium therapy is well described. Bauer deals with constitutional conditions. He gives an elaborate discussion of the problems of heredity, and this is followed by an account of the rôle of the endocrine glands and of diabetes in relation to surgery.

The second Part published, pp. 386, with 215 figures and 14 coloured plates, will form a section of Vol. III. It deals with the lymph-glands, the breast, the glands of internal secretion, and the pituitary. Neupert takes the first of these subjects, and gives a thorough account of the lymph-gland system from the structural and pathological points of view. The anatomical distribution of the glands is very briefly described, and presumably will be included in various special subjects. The coloured figure in this article is unaccompanied by any legend, and it is not easy to find reference to it in the text. The surgery of the breast is by Klose and Sebening, and constitutes a most valuable and complete monograph, with 13 coloured plates in addition to 122 figures in the text. The anatomy and pathology are particularly well described, and the illustrations are complete and artistic. The debatable question of the spread of cancer is hardly touched upon. It is said to spread chiefly by the lymphatics and to elect the vertebral bodies and neck of the femur as favourite sites for metastasis, but the facts of perilymphatic fibrosis, the distribution of the cutaneous nodules, and the theory of permeation are not mentioned. It surprises the English reader to find a complete account of Warren's operation

for removal of cysts, with four figures (source not mentioned or acknowledged), but no mention of Handley's name or work.

The chapters on the thyroid, parathyroids, thymus, and adrenals are by Sudeck. They contain an excellent and well illustrated account of the histology of various thyroid affections and also of all the stages of exophthalmic goitre and its treatment. The special characters of metastasis in thyroid tumours are mentioned but very briefly, and are not illustrated at all. Kocher's work dominates this section, with notable omissions of some other authorities. Oehlecker deals with the surgery of the hypophysis, and gives a short but complete account of its anatomy, functions, and the methods of surgical approach. In the latter the transfrontal route is mentioned as the best, but it is the temporal route which is illustrated.

A Text-book of Operative Orthopedics. By A. STEINDLER, M.D., F.A.C.S., Professor of Orthopedic Surgery, State University of Iowa. Royal 8vo. Pp. 403 + xv, illustrated. 1925. London: W. Appleton & Co. 30s.

THE author's name having become well known as the originator of a special operation for claw-foot, his book will attract considerable attention. In the early parts of the volume, especially the section on tendon transplantation, our expectations are fully realized; but in the later chapters, and notably in those dealing with reconstructive bone operations, we are disappointed. The aim and general design of the book are very good. The author sets out to examine, first, the rationale of each operation as judged by its physiological and mechanical basis, giving special weight to experimental evidence. The clinical aspect is mentioned very briefly. The description of the operative technique and steps of the operation naturally occupies the biggest share in the book, and this is illustrated freely by diagrams, which are clear though sometimes crude, and by reproductions from original papers. There is an attempt to give a statistical account of operation results. This is certainly an excellent idea, though it is, of course, open to all the fallacies which are inevitable in trying to express complicated ideas in terms of simple numbers.

Each chapter ends with a list of the chief papers and references, and this seems to be well chosen. It has no claim to completeness, but it does serve to help the reader to get in touch with the principal authorities on any subject.

The most serious criticism of the book is that it attempts to deal with far too much in a very short compass. If a book of this size had been devoted to what used to be called orthopædics, i.e., the surgery of deformities, the subject could have been adequately treated. But subjects such as spinal-cord surgery, and the surgery of fractures and joints, are also included, and these sections would require a much larger space to do them justice. Again, in order to achieve completeness, methods of doubtful value, e.g., cinematization of stumps and ramisection, are described, but not with sufficient detail to serve as a useful guide. It is undoubtedly a good book, but in future editions we venture to suggest that the author will have to choose between dealing thoroughly with a small subject or else greatly enlarging the work.

The Surgery of Pulmonary Tuberculosis. By JOHN ALEXANDER, M.A., B.S., M.D., Assistant Professor of Surgery in the Medical School, University of Michigan, etc.; with Introductions by HUGH CABOT, C.M.G., LL.D., M.D., F.A.C.S., Professor of Surgery and Dean of the Medical School, University of Michigan, and EDWARD R. BALDWIN, M.A., M.D. Medium 8vo. Pp. 356, with 53 engravings and 12 plates. 1925. London: Henry Kimpton. 21s. net.

THE bibliography at the end of Dr. John Alexander's book contains 500 references to contributions on the subject of the surgical treatment of pulmonary tuberculosis, for the seven years 1918 to 1925. This is a clear indication of the extensiveness of the work which has been done and of the interest which has developed in this subject in recent years. The perusal of such a wealth of literature presents a formidable task even to the man keenly interested in the subject; owing to time and language difficulties, it is an impossibility to many. Dr. Alexander has therefore rendered a very great service to physicians and surgeons by extracting from this literature

all the salient facts. He has, in connection with each mode of treatment, brought together in a concise form the views of all those who have had practical experience of the methods. Where there is divergence of opinion, the various arguments have been very fairly compared and judicially summed up. For this reason the book is essentially one to be read and studied by the physician and the surgeon. The former will obtain from it an extremely fair knowledge of the immense value of surgery for pulmonary tuberculosis, but will at the same time learn the limitations. For the surgeon there is, in addition, a wealth of information as to the technique of the various operations, and variations of the same type of operation.

Having traced the evolution of thoracic surgery, and gone at length into the indications and contra-indications for surgical intervention, the author deals with all the modern operations: extrapleural paravertebral thoracoplasty, phrenicotomy (under which heading is included exairesis or evulsion of the phrenic nerve), extra- and intrapleural pneumolysis, and drainage of cavities, tuberculous effusion, and empyema. Artificial pneumothorax is not included, but there is a chapter on the comparative values of artificial pneumothorax and thoracoplasty. This comparison is extremely fair, and is of great assistance in view especially of the increasing tendency on the part of some of the German surgeons to advocate thoracoplasty.

The author shows of what great value the various surgical measures have been in saving lives which would otherwise undoubtedly have been lost. He emphasizes, and rightly too, how erroneous is the general impression of thoracoplasty; that the 'hazy notion' of the terrific shock and mortality, of the pain and post-operative deformity, is in no way based upon fact (p. 19). He has collected a total of 1159 thoracoplastic operations. The percentage results are: Cured, 36·8; improved, 24·4; living but unimproved or worse, 5·25; dead from causes directly or indirectly connected with operation, 14·1; dead from causes not directly connected with operation, 33·5. It must be remembered, he points out, that all these were advanced cases and all of them had had (with few exceptions) a prolonged course of sanatorium treatment, and possibly artificial pneumothorax treatment, which had failed to arrest the disease.

Dr. Alexander points out what a very small number of those patients who are suitable are offered the chance of surgical treatment, and of the possibility therefore of 'cure' or improvement. The great object of his book is to bring to the notice of those who have to advise these sufferers from pulmonary tuberculosis the possibilities of happy intervention—possibilities which are at the present too generally neglected.

It is interesting to note that "this monograph has been awarded the 1925 quinquennial Samuel D. Gross prize by the Philadelphia Academy of Surgery".

Selected Papers: Surgical and Pathological. By F. T. PAUL, D.Sc. Liverp. (Hon. Caus.), Ch.M., F.R.C.S., Consulting Surgeon, Liverpool Royal Infirmary. Demy 8vo. Pp. 284, with 23 plates. 1925. London: Baillière, Tindall and Cox. 15s.

A COLLECTION of published papers by Paul, of Liverpool, comes to this Journal for the purpose of review; but it would seem that they should be rather the subject for appreciation than for a review, for they indicate the feeling of the surgeons of Liverpool to one of their oldest colleagues. No better compliment or mark of respect could be paid to a man than such a gift from his own associates.

Frank Thomas Paul was born on Dec. 3, 1851, at Pentney, Norfolk. He was educated at Yarmouth Grammar School and Guy's Hospital, where he gained an exhibition and held the office of house physician. He qualified in 1873, and in 1878 became a Fellow of the Royal College of Surgeons of England. He was appointed Surgeon to the Royal Infirmary, Liverpool, in 1890, and also held the office of Dean of the Medical Faculty and Professor of Medical Jurisprudence in the University of Liverpool. An all-round man, a careful observer, interested in many things, Paul has made valuable contributions on many topics of surgery and pathology, but his name will ever be associated with the surgery of the large bowel. Undoubtedly it is due to him that the surgery of the colon is what it is to-day, and all students are familiar with 'Paul's tubes'. We would like to associate ourselves with the Liverpool surgeons in our congratulations to Mr. Paul.

La Pratique chirurgicale illustrée. By VICTOR PAUCHET. Imperial 8vo. Vol. VII, pp. 256, with 188 illustrations; Vol. VIII, pp. 250, with 265 illustrations. 1925. Paris: Gaston Doin. 30 francs net, each volume.

THESE further sections of Pauchet's work are very similar to former volumes. They contain a miscellaneous collection of operations performed by the author, and are illustrated by somewhat diagrammatic drawings of the appearances seen by the artist during the actual course of the operations. In Vol. VII the first two chapters are written by other surgeons: the treatment of facial wrinkles by Virenque, and the technique of staphylorrhaphy by Victor Veau. The obliteration of the wrinkles of age by surgery will not appeal to Englishmen, but the chapter on cleft palate is interesting, as some ingenious instruments and suturing methods are advocated. It is recommended to suture the palate before the end of the first year unless the cleft is very wide. Pauchet writes a clear description of an extensive operation for a malignant thyroid, after which the non-operative treatment of fractures of the upper extremity is dealt with. Some of the methods differ from ours, but they always appear logical, though the apparatus seems rather cumbersome on occasion. The author recommends leaving the head of the humerus in its dislocated position when a fracture of the anatomical neck occurs in association with a dislocation of the shoulder.

It is not surprising to find Pauchet returning again to the subject of gastric surgery both here and in Vol. VIII. Much of the ulcer surgery is dealt with in former volumes, but cancer of the stomach is more fully described than elsewhere. Fifty per cent of the cases coming to him are suitable for surgery. The mortality after operations for cancer of the stomach is 20 per cent, but 10 per cent live more than ten years. At the end of Vol. VII is rather an original way of treating a perforated duodenal ulcer. In Vol. VIII methods of skin-grafting are described. There is a good chapter on goitre surgery, with remarks on the use of X rays in treatment. Salivary fistulæ, umbilical hernia, undescended testicle, and excision of the colon form short sections. The operation for intestinal obstruction seems to require too much exposure of the intestine, and too much manipulation, to be entirely satisfactory. Excision of cancer of the prostate forms a very interesting chapter and is well worth perusal.

This atlas of surgical operations is unique. The illustrations are wonderfully clear. The author's activities are many-sided, and surgery in his hands is evidently pushed to its limits. The books form an extraordinary record of one man's work.

Traitement des Fractures et Luxations des Membres. By JACQUES LEVEUF, Chir. des Hôpitaux de Paris, and CH. GIRODE, P. MORNARD, and RAOUL MONOD, Chefs de Clinique à la Faculté de Médecine de Paris. With a Preface by Professor PIERRE DELBET. Crown 8vo. Pp. 464, with 247 illustrations. Paris: Masson et Cie. Fr. 25.

THIS is an interesting and valuable book, because, emanating from a group of younger Paris surgeons, and inspired by the idea of Delbet's master mind, it may be taken as a fair representation of the French hospital practice.

It is divided into two parts. The first deals with non-operative methods, manipulation, splinting, and after-care. The details of the apparatus used differ from those we are accustomed to, though the main principles are the same. Delbet's traction apparatus for fractured femur is given great prominence. To us it seems difficult to believe that this apparatus can give any precision, and it must require much experience before it can be used safely.

The second portion of the book is devoted to the open operations for fractures. In it the teaching and practice of Lambotte is followed closely. The various methods of Parham's bands alone and with plates are also given some prominence. Apparently the French surgeons are entirely satisfied with the metallic fixation of broken bones. They appear to be almost unaware of the use of bone nails, screws, pegs, or grafts. For example, in fracture of the neck of the femur a long carpenter's screw is depicted, whereas the bone nail, which gives a firmer fixation and becomes incorporated in the tissues, is ignored. The book, in regard both to text and illustrations, is a model of brevity and clearness.

Praktikum der Chirurgie. By Professor Dr. O. NORDMANN. Third edition. Royal 8vo. Pp. 796, with many illustrations. 1925. Berlin: Urban and Schwarzenberg. Sewed, M. 33; bound, M. 37·50.

THIS is a large book of a kind a little different from any we have in this country. The author does not give systematic accounts of the different diseases, but confines himself to accounts of the clinical features and diagnostic difficulties met with at the bedside. Treatment which is ordinarily carried out by residents, and emergency operations, are fully described, but other major operating is indicated only. It is interesting to note that defibrinated blood is used as often as citrated blood for transfusion. We observe that Murphy's drip method of proctoclysis is referred to as Katzenstein's, and the procedure described ignores Murphy's insistence on the fact that the conducting tube should be unobstructed by any clamp. In the section on appendicitis the author rightly, we think, recommends that, in general, drainage only of a localized abscess should be done. With regard to the habit of closure of the abdomen when diffuse peritonitis is present, he adopts an attitude of caution, believing that it should not be done as a routine, but only in carefully-selected cases. Cholecystectomy is performed from the fundus towards the neck, a method which has a good deal in its favour. After any laparotomy, if signs of diffuse peritoneal infection occur, Nordmann says that any treatment may be carried out except re-laparotomy, which is invariably followed by death. Altogether the book seems a reliable guide for the student and emergency surgeon.

Diagnostik mit Freiem Auge. By Dr. EDUARD WEISZ. Foreword by Professor Dr. FRIEDRICH KRAUS. Second edition. Royal 8vo. Pp. 178, illustrated. 1925. Berlin: Urban and Schwarzenberg. Unbound, M. 7·20; bound, M. 8·70.

THIS is a monograph of 178 pages which might easily have been compressed into a smaller compass. It deals solely with a method of diagnosis called ectoscopy, which consists in the careful observation of the respiratory movements that can be seen in the intercostal spaces, neck, and abdomen. Several ways of carrying it out are described: inspection in quiet breathing, inspection on quicker but not deep breathing, the speech and sniffing phenomena. There is no doubt that the author has done a service by calling attention to the necessity for accurate observation, which indeed all the older clinicians insisted on; but whether this method is or will be of great use may be doubted. He affirms that the eye becomes skilful with training, and can observe slight movements which at first were undetectable, that the lower limits of the lungs can be seen, and that help is gained in many thoracic conditions and in some abdominal diseases. It is careful and interesting work, the worth of which only a trial can demonstrate.

Methods in Surgery. By GLOVER H. COPHER, M.D., Instructor in Surgery, Washington University School of Medicine; Clinical Assistant to Barnes Hospital; Surgeon to the Washington University Dispensary and St. Louis City Hospital. Crown 8vo. Pp. 232. 1925. London: Henry Kimpton. 14s. net.

THIS book is a guide for house surgeons, more particularly those working in three Washington hospitals. Its interest lies in the insight it gives into the organization of these hospitals, especially the arrangements for the keeping or consultation of clinical notes, for obtaining radiographic, chemical, and other examinations, and the instructions given to the residents. Some of these latter appear strange to us, as, for example, the admonition not to wear their hats in the laboratories and not to ask fees from the patients. The different diets classified according to their calorific value are useful. The directions given for pre- and post-operative treatment are rather lacking in detail. There is nothing very striking about the book.

Guy's Hospital Reports. Vol. LXXV, No. 3, July 1925. Edited by ARTHUR F. HURST, M.D. Royal 8vo. Pp. 124. London: Wakley & Son (1912) Ltd. Annual subscription, £2 2s. net; single members, 12s. 6d. net.

THE reports of Guy's Hospital continue to maintain their interest, which is remarkable considering the very large number of medical journals that are published throughout

the year. To those interested in Keats there is a delightful article which will appeal, entitled " Keats as a Medical Student ", written by Sir William Hale-White. There are two articles on œsophageal diverticula which are of surgical interest; they refer to those rare cases of pouching of the lower end of the œsophagus.

Chirurgische Operationslehre. By Professor FRIEDRICH PELS LEUSDEN. Fourth edition. Royal 8vo. Pp. 832, with 771 illustrations. Berlin and Vienna: Urban and Schwarzenberg. Sewed, M.18; bound, M.21.

THIS is a text-book on operative surgery similar to those published in most languages. As it is a paper-covered book, written in German, and does not contain any specially new matter, it does not appear likely to commend itself to the English reader.

Scritti medici in Onore di Antonio Carle. In two volumes. Royal 8vo. Pp. 756 and 785, illustrated. 1925. Bologna: Licinio Cappelli. Each, Lire 20.

VOLUMES XII and XIII of the *Italian Archives of Surgery* are devoted to contributions from a great number—more than a hundred—of Italian surgeons, as a tribute to the work and teaching of Antonio Carle. Many of the most famous names in contemporary Italian surgery are included, and the 1500 pages together give a brilliant picture of their present-day activities. Not all the articles are of great importance; some are little more than careful records of remarkable cases; some of the more important are not entirely new—for example, the beautifully illustrated report of his experimental work on acute pancreatitis by Calzavara; but as a whole it is a splendid witness, by pupils and friends, of the esteem in which Carle's thirty years of teaching is held.

It is impossible to review, even cursorily, more than a fraction of the contents. Professor Donati contributes to the study of traumatic shock an experimental investigation into the method of action of toxic substances derived from contused tissues. Lattes throws doubt on the limitation of blood-groups to four. There are several studies of grafts of formalin-fixed tissues. Professor Purpura writes on blastomycosis. Of two or three papers on echinococcus, one, Nassetti's, suggests a method of treating the cyst wall similar to Matas' treatment of aneurysm. Palmieri describes a method of indirect X-ray irradiation which permits 'cross-fire' attack from a single source. There are reports of investigations into the presence of organisms in the bone medulla of simple fractures, and in the epiphyses (Fiori and Uffreduzzi). Cranial and cerebral surgery are represented by, among others, Bufalini and Solaro. Bertolotti prints some beautiful radiograms of cranio-pharyngeal tumours. Torraca records a case of total necrosis of the mandible, and de Gaetano discusses the operative treatment of ankylosis of the jaw.

In the second volume Parlavecchio indicates the technique of partial and complete resection of the sternum; Anzilotti deals with the cure of bronchial fistulæ, insisting that complete liberation of the lung from the chest wall is the *sine qua non* of success. Vespignani, who writes on the direct imprint of the left costal arcade on the stomach, and its importance in reading opaque-meal appearances, is not very convincing. Costatini figures a plastic method of overcoming the difficulties encountered in excising gastric ulcers near the cardia. Gastric function and peptic ulcers are the subjects of half a dozen papers; the biliary tract and function of another group; in the renal field, amongst others, Stropeni describes experiments on the influence of decapsulation on the renal circulation. Muscatello relates a case, said to be the first, of inveterate horizontal luxation of the humerus. Durante records a case of periarterial sympathectomy of the posterior tibial after failure of a similar operation on the femoral; Scalone (in Vol. I) describes his technique for this operation; his diagrams are, to say the least, optimistic. There are several useful orthopædic contributions.

The standard of the work is high, and the field covered vast. Professor Carle must indeed feel himself honoured.

Peritoniti acute. By DOTT. V. PUCCINELLI. Preface by Professor RAFFAELE BASTIANELLI. Imperial 8vo. Pp. 281, illustrated. 1924. Bologna: Licinio Cappelli. Lire 35.

THIS book is honoured by a preface from the pen of Professor Bastianelli, a full record of whose cases from 1900 to 1923 appears in the second part.

The first part, 92 pages, is devoted to a review of recently expressed views from all sources on the physiology, pathogeny, histology (well illustrated by photomicrographs and drawings from the author's specimens), pathology, symptomatology, prognosis, and treatment of peritonitis in its various forms. There is also a short section describing new experiments on absorption from the peritoneal cavity.

The second, statistical, part is of course valuable, for it contains short notes of 1005 operation cases, of which 484 were due to war wounds. Peritonitis due to perforation of the appendix shows a mortality of 27·4 per cent; but it must be remembered that cases as far back as 1900 are included, and that patients still arrive at hospital much too late. Professor Bastianelli deplores the ill success of all his efforts to educate the public and the profession in the supreme importance of promptitude in resorting to operation. Perforated peptic ulcer, mortality 37 per cent; typhoid perforation, 73 per cent; perforation of tuberculous ulcer, 63 per cent; 'diplococcal' peritonitis, 5 out of 7 cases; puerperal peritonitis, 54 per cent; gynæcological, i.e., chiefly salpingitis, 29 per cent: this gives a total mortality of 42 per cent in 412 consecutive cases of peritonitis other than that due to wounds.

The third section of the book, more than sixty pages, is devoted to bibliography.

Operative Surgery. By J. SHELTON HORSLEY, M.D., F.A.C.S., Attending Surgeon, St. Elizabeth's Hospital, Richmond, Va. Second edition. Imperial 8vo. Pp. 774, with 666 illustrations. 1924. London: Henry Kimpton. 52s. 6d. net.

THE first edition of this work appeared in 1921, so that the need for a second within three years speaks well for its popularity. As pointed out in the preface of the first edition, the author does not intend that the book should cover the whole field of operative surgery. The operations which are described are those which he is in the habit of performing, or are those which he considers to be best suited to the condition needing surgical interference.

A special feature of the present edition is the inclusion of interesting new operations which, so far as the author is aware, have not hitherto appeared in either text-book or monograph. These operations are: the lymphaticostomy of Costain, Storkey's operation for innervating paralysed muscles, Finney's pylorectomy, Graham's pulmonary lobectomy, Cutler's valvotomy for mitral stenosis, the operation of Coffey and Brown for angina pectoris, Frazier's chordotomy, and Kerr's method of intestinal resection. All of these operations are carefully described, and form a valuable addition to the book.

The work has been brought thoroughly up to date, and maintains the high grade of excellence displayed in the first edition. We can cordially recommend a perusal of its pages to all those who are desirous of acquainting themselves with the best methods of American operative surgery.

Chirurgie de l'Appareil urinaire et de l'Appareil génital de l'Homme. By PIERRE DUVAL and J. GATELLIER. Sixth edition. Crown 8vo. Pp. 284, with 310 illustrations. 1924. Paris: Masson & Cie. Sewed 12 fr., bound 15 fr.

THIS book is one of the series of Précis de Technique opératoire; it is the work of the Prosectors of the Faculty of Medicine of Paris, and represents their teaching of operative surgery. It contains 281 pages and is a miracle of conciseness; in addition to the 310 illustrations, there is an excellent description of the operations commonly performed on the kidney, the ureter, the bladder and prostate, the urethra, and the testicle—and the book can be comfortably carried in one's pocket. The numerous illustrations are in black and white, and not only show the various operations, but in some cases depict the anatomy of the parts concerned; they are extremely well done, and are a real help to the understanding of the text.

As the work is intended to be a handbook for a course of operative surgery, there is no clinical information, but as a manual of surgical technique for a special department of surgery it is very good, and we can recommend it to our readers.

La Secrezione gastrica. By Dott. ANTONIO CIMINATA. Royal 8vo. Pp. 247, illustrated. 1925. Bologna: Licinio Cappelli. Lire 35.

THE admirable series of monographs published by the firm of Licinio Cappelli at Bologna deserves special mention for its excellent typography, the high standard of its illustrations, and its freedom from errata, as well as for the character of the work itself. This volume is a good example of the work being done in the Italian universities. In the main it is a record of a large series of experiments on dogs, in continuation and confirmation of the work of Heidenhain and of Pawlow, which is described and discussed, together with other work on the same subject, in the first section of the book. The author gives a full account of his method of making a small attached but separate stomach for investigation of the secretions, and of the very numerous experiments conducted.

Among the results he observes are the following: a direct relation between the percentage of water in the food and the quantity of juice; a direct relation between quantity of succus and period of latency; the period of maximum secretion and the duration of secretion vary with the type of food; water, by itself, stimulates a flow, but it is scanty and of brief duration; the degree of acidity corresponds to velocity of secretion, not to type of food; peptic value corresponds to the type of ingesta.

He concludes that the process of secretion considered as a whole is an adaptation of glandular activity to the qualities of the food; that most foods contain in some degree chemical stimulants of secretion, which are developed in the earliest stage of digestion; the first flow of juice necessary to start their production is occasioned by psychic reflex—appetite, in fact—and perhaps by water itself. The pyschic reflex reaches the stomach by the vagi; the subsequent chemical stimulus, equally 'nervous', acts, however, directly on the plexuses in the stomach walls. Fats inhibit chemical stimuli. Acidity does not vary directly with different foods; the degree is determined by the amount of neutralizing mucus, and that depends on the rate of flow of succus; 'hyperacidity' merely means a not-yet-neutralized succus, a failure of equilibrium. It is extremely doubtful if succus secretion is in any way influenced by substances reaching the cells by the blood. There is a bibliography.

Megacolon congenito. By Professor GUALFARDO TONNINI. Imperial 8vo. Pp. 243, illustrated. 1924. Bologna: Licinio Cappelli. Lire 20.

THIS monograph covers the whole history and literature of Hirschsprung's disease and its allied conditions. It gives a résumé of 63 cases from Italian publications besides details of a personal case. It suffers, like so many similar Italian books, from a plethora of diverse descriptions and opinions, amid which it is difficult to discover what are the author's own conclusions; there is evident reluctance to assess the value of the diverse views. Probably the truth is that congenital megacolon is not one condition but many; and if this book contributes, as it should, to their disentanglement and discrimination, it may be possible to standardize treatment. Very little is said here, except in the author's own case report, about radiological investigation, yet the condition is pre-eminently one for such study; the passage of an opaque meal and the flow of an enema ought to reveal a good deal as to the site and character of the 'obstruction', and give valuable information for determining the type of operative treatment best adapted to cure. There is an extensive bibliography.